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XLIX.

TEMPUS FUGIT.*

ROSS HALL SKILLERN, M. D.,

PHILADELPHIA.

Did it ever occur to you that when time flies it takes something with it, and when it does that something is gone—gone? What unutterable sadness that word implies! Say it to yourself when the day is over, in the quiet of your study; say it to yourself in the peacefulness of the cemetery where one of your loved ones lies sleeping; say it when you think of men whose hands you clasped at a recent meeting, such as Charles Richardson, Macuen Smith, and you will realize its meaning as you have never before. As we grow older our friends pass on and are gone. They who counseled and advised us, one by one drop away as we journey on, and yet our own journey does not seem a long one. But enough of these melancholy reflections. Let us consider our subject from a less personal, if not more interesting angle, and discuss in a more or less disjointed manner some of the thoughts

*Presidential Address, American Laryngological, Rhinological and Otological Society, Atlantic City, May 28, 1930.

that the passage of time has impressed upon our perception and memory.

In the Metropolitan Museum, in New York, there is a papyrus which is said to date from the time of Cheops, about 3700 B. C., and concerns the preparation of a drug that is burned and the vapor inhaled for certain throat troubles. This, to my mind, represents the first known allusion to our specialty. Many hundreds of years later, about 930 B. C., in an age which seems almost nebulous, Homer tells of certain of the Greeks who were skilled in curing special affections. Among these the throat is mentioned. Thus sprang into being the laryngologist, for our word larynx is derived from the Greek larynz, meaning upper part of the windpipe. Herodotus, the Greek historian, writing in the fifth century B. C., states that among the early Egyptians no doctor was permitted to practice except in his own branch, showing that specialists existed even at that remote period. From our meager knowledge, this would seem to show that when Europe was in the throes of barbarism and Greece had not yet appeared as a civilized state, the Egyptians had perfected certain arts of civilization which in some respects have not been equaled to this very day. We would know infinitely more about the ancients and their manners and customs had it not been for the intolerance of succeeding ages, who could see in their ignorance and fanaticism little that was worth preserving in the archives of the conquered.

Intolerance has been the greatest enemy to advancement since the beginning of civilization. This is particularly true in respect to culture and learning. Consider for a moment the vast stores of treasures that would have been at our hand had not this obtained. Begin, for example, with the Alexandrian libraries, the first great destruction occurring when one of Caesar's ships became afire and spread to the city; the next one was destroyed by fanatical Christian Archbishops, and finally, when the city was conquered by the Mohammedans, word was sent to Caliph Omar to learn what disposition should be made of the contents of the libraries, and he replied: "If they agree with the Koran, they are not needed; if they contradict it, they are false—so in either case destroy them."

These priceless scrolls were used as fuel to heat the caldariums of their public baths and are known to have answered this purpose for over six months. Despite this, however, it is astonishing the records that have come down to us from early Egyptian and Babylonian times in the papyri and stone tablets. For example, in the Ebera papyrus, that was written about the time Moses was tending his flocks at the foot of Mount Horeb, many recipes are mentioned that called for certain drugs which are in common use today. Despite, however, the primitiveness of this early Egyptian medicine, nevertheless, we all still use an abbreviation which was used by Chaldean physicians before our alphabet had been invented. I refer to the ℞ with which we begin our prescriptions. I was taught this signified recipe or take of, but further investigation shows that it may be traced to a pagan symbol invoking Jupiter or some such similar deity that was worshiped by the Chaldeans.

The Assyrians and Babylonians have also left us information of their knowledge of medicine on the many clay tablets which have been preserved and are from time to time unearthed. The symbol of the serpent seen on our Caduceus descended originally from the Babylonians and has since erroneously been affiliated with Hermes, who in reality had nothing whatever to do with medicine. It is highly probable that many of the manuscripts lost in the fires of the Alexandrian libraries were written in Egyptian, Greek, Latin and perhaps ancient Hebew, of which no copy or translation remains. Specialties certainly existed at that time, and who knows, if some of these old manuscripts were still extant, that certain things of which we still remain in ignorance may not have been clarified so that functions may have been explained which now are no longer understood. Bringing this down to our own specialty, the efficacy of our treatment of diseased conditions is in direct ratio to our understanding, and appreciating the functions of the particular part affected—thus, in the era just before I became interested in rhinology it was the common custom to resect the entire inferior turbinate for interference with nasal respiration. Now that we appreciate the true function of these bodies which moisten and warm the inspired air, no one thinks for a moment of performing this operation, thereby rob-

bing that side of the nares of its most useful function, at the same time causing that unfortunate condition known as a dry nose. Now all this has come about because the true functions of these turbinate bodies have been recognized. Well, then, how about the sinuses? That they have or have had a function no one will deny, but of what use are they to the human individual? Many theories have been advanced: to lighten the bones of the face, to give resonance to the voice, to help moisten and heat the inspired air; but while they probably take part to a minor extent in all of these, nevertheless, their chief function apparently remains unrecognized. Unless we know these cavities in health, how can we know them in disease?

We are very much in the position of the agnostic, who contends that true knowledge does not exist, because all knowledge is relative and uncertain. This seems to doubly apply to the sinuses, for in the first place they are never morphologically alike in any two individuals, therefore, they cannot in every case show exactly the same symptoms under like pathologic conditions. Under these circumstances, we can only depend upon our relativity of knowledge, either as revealed in perception of sensible phenomena (such as subjective and objective symptoms) or as shown by the element of error in abstract conceptions, such as hypothetical assumptions. These facts, to my mind, are responsible for the existence of the two schools of treatment in sinus conditions: the Conservatives and the Radicals. The Conservatives, because in the widespread and extensive resections they see the possible functions of these cavities destroyed, and, even though the infection be temporarily alleviated, the ultimate condition, dependent upon a locus minor resistentia, continues to give more or less trouble during the whole remaining life of the individual. The Radicals, on the other hand, contend that the infection must be eliminated root and branch in order to bring about a complete and permanent cure. Now it seems to me the real truth lies between these two viewpoints, and the reason that both of these schools hold to their tenets is primarily due to our own faults and actions. Our faults, because we have never agreed as to the amount of mucosa that should be removed after radically opening a sinus, and our actions, because after opening we either

remove too much tissue or leave behind areas which should have been eradicated.

Now, what was the basic cause of this disagreement? Because the Conservatives and Radicals could not agree upon the amount of tissue to be removed in a given condition, the former averring that if the worst of the diseased mucosa was curetted and free drainage established Nature would speedily bring about resolution in the part that was left behind; while the Radicals just as emphatically stated that all tissue that was permanently pathologically affected could under no circumstances return to normal, therefore should be removed. It would seem to me much is to be said on both sides, the basic principle of the entire contention lying in our inability to reach an agreement as to just what condition the mucosa must be in as to indicate its removal. The whole matter lies in the personal opinion of the attending sinuologist, and as long as our interpretations of pathologic symptoms differ, just so long must we expect to have diversities of opinion as to the operative procedure indicated in any particular case. If we could bring it about that the teachers in our graduate schools would agree to divide the macroscopic appearance of the diseased sinus mucosa into three types:

Type 1. Where the pathologic condition of the mucosa was so altered that removal in whole or in part was indicated without further discussion.

Type 2. Where the mucosa was diseased but it was questionable as to its removal.

Type 3. Where the mucosa was so little changed that ventilation and drainage would unquestionably be all that was necessary for complete restitutio ad integrum.

In this manner there would be only one type that would permit of any discussion, but the personal element regarding interpretation of the mucosa would so enter into this as to make it impossible to come to an understanding just what type 2 really embraced; therefore, I fear we will always be divided into Conservatives and Radicals. They say as time goes on, one who operates frequently grows more and more conservative. I have often had occasion to observe this apparent trend in many of our prominent surgeons, but such a statement seems to obscure the

point. They do not become more and more conservative, but more and more careful. It is also true that unless we continually watch ourselves we are apt to become the slaves of custom.

How prone we are to follow certain procedures or lines of treatment, because it is customary, even though there may not be the faintest rhyme or reason why such should be followed, nor, if asked, could we give a satisfactory explanation. This pertains not only to medicine but even to our life habits. The universal custom is to don new raiment on Easter Sunday and stroll out to see and be seen, but how many of us could give an adequate explanation why such procedure should obtain on a certain given Sunday of that year? As a matter of fact, the precedent has been well established, for in the early part of the fourth century Constantine, the first Christian Emperor, being fond of gaudy ostentation, caused Easter to be celebrated with great pomp and circumstance, and commanded everyone to appear in attractive new apparel. This custom has come down to us through the ages, although the inception and real significance have long since been forgotten. Bringing this up to date, as far as custom is observed, one is reminded of the present divergence of viewpoint between certain of our ophthalmologists and the sinuologists in the treatment of retrobulbar neuritis. When this condition occurs in an individual, with comparatively rapid loss of vision, the ophthalmologist has been laying too much stress on blood counts, Wassermanns, bacteriologic cultures, etc., thereby wasting valuable time as far as the patient's sight is concerned; whereas, the sphenoid should have had instant attention. Recoursing at this time to the expediency of the laboratory in order to determine a possible but hardly probable etiologic identity represents but dilatory tactics and in reality the admission of our own limitations in the knowledge to successfully cope with such pathologic problems. This time is the vital moment for instant decision; either the mailed fist is to be applied, being guided purely from the sense of rhinologic intention, or, seeking the lines of least resistance, valuable or even vital time (ophthalmologically speaking) is lost in the search for the oft too suspected spirochete.

We are apt only to acquaint ourselves partially with certain knowledge and then feel we are in mastery of the important facts

and details. For instance, every schoolboy knows that Prometheus was a mythologic personage who was by the order of Zeus bound to a cliff where vultures continually fed upon his liver, but how many of these schoolboys knew or cared what became of him, most of them thinking he ultimately perished from this torture, where, as a matter of fact, he was finally released and restored to Jove's good graces. The same state of mind as that of the schoolboy might easily occur with us, in, we will say, the treatment of certain cases in which we have been particularly successful. Suddenly a case comes which seems to resist and withstand our usual therapeutic procedures which have hitherto been so satisfactory. To ease our conscience we advance all sorts of explanations, but deep down in our hearts we realize that in this particular case something is different from the others which we do not understand. Now, to the thinker, is the time to disregard the usual conventionalities, and instead of accepting the inevitable, to exhaust every possibility until the proper clue is obtained and the proper remedy applied. It is this alone that spells the difference between the master and the man.

But my time has been consumed and perhaps to many of our younger members the theme of this discourse has been somewhat melancholy, but those of you who, with me, have been members of this society for more than two decades, with memories of those twenty-odd years of the passing realities gone, never to return, can look at one another and indeed say, Tempus Fugit.

L.

THE RELATIONSHIP BETWEEN INFECTIONS OF THE UPPER RESPIRATORY TRACT AND PEDIATRIC CONDITIONS.

L. W. Dean, M. D.,

St. Louis.

When I was asked to speak to you I thought that I would discuss the relationship between internal medicine and pediatrics and infections of the upper respiratory tract. I soon found that this subject was too vast, so I have limited my remarks to the relationship between infections of the upper respiratory tract and pediatric conditions.

I trust that the pediatrists with whom you come in contact are good otolaryngologists. Those whom I know are excellent ones. Ignorance of this subject is the one thing that prevents us from rendering the very best service to that class of patients who are directly under the care of others.

Sixty per cent of all human ills are located above the diaphragm. Many located below the diaphragm are associated with infections above, so really in diseases of children it is impossible to dispense with the services of the otolaryngologist.

Naturally the pediatrist and the otolaryngologist will work together; there will be a conference in each case. The pediatrist can help us with our patients as much as we can help him. He is the best judge as to the general condition of the patient. Certainly if a child has a mastoid which is in itself a typical surgical case the child should not be operated upon if the pediatrist says the shock of the operation will cause death. In sick infants with mastoid infections it requires the combined judgment of both to render to the infant the very best service.

The otologist must take into consideration the future hearing of the child, the danger of meningitis, of thrombophlebitis. The pediatrist thinks of the probability of getting the child in condition so that the drainage can be done in greater safety.

Certainly when an infected infant's ear is responsible for the serious general condition of an infant, an infected mastoid, however bad, should not always be drained until the general condition of the child is improved.

When the drainage operation is done the pediatrist must prepare the child for the operation and care for it afterwards. In short, I am convinced that this is advisable in all operations in infants and children.

Common colds produce decided upsets in infants. In infants and in children they are of importance because of the danger of complications—otitis, sinusitis, tracheobronchitis. I doubt very much if treatment hastens the termination of the cold. However, we can usually prevent complications and give the patient relief. The essential things are diet and proper hygiene. Locally one-half per cent ephedrin in oil may be of benefit.

In infants and children with common colds, instead of terminating in three or four days, the nasal symptoms may diminish but fever persists. Such patients may have an otitis or a sinusitis with or without tracheobronchitis. If tonsils or adenoids are present they are always under suspicion. If they are out and there is an acute cervical adenitis you are almost sure to have a nasal sinusitis. If so, your patient will not do well until this trouble is controlled.

Nontuberculous and nonsyphilitic cervical adenitis, acute or chronic, always suggests in children infected lymphoid tissue in the pharynx, nasal sinusitis or infected teeth. The treatment is that of the infection about the upper respiratory tract.

In tuberculous cervical adenitis the infection is usually through the tonsils, teeth or walls of the pharynx. Frequently the tonsils and adenoids are tuberculous. Unless the tonsils and adenoids are undoubtedly normal and they can be excluded as portals of infection they should come out. The teeth and any pharyngitis present should be treated. The condition of the nasal sinuses should be investigated and any pathologic condition eradicated.

Much has been written recently about the ocular complications of infections about the upper respiratory tract. In infections of the uveal tract and lesions of the optic nerve, in my mind, infec-

tions of the tonsils take precedence over that of teeth and sinuses as contributory factors. The nasal sinuses are least important in spite of the close anatomic relationship of the posterior ethmoids and sphenoids to the optic nerve. This does not mean that the sinuses are ever to be overlooked as sources of infection. In orbital cellulitis and edema the infection of the nasal sinuses ranks first in importance as etiologic factors.

In chronic bronchitis and bronchiectasis the first step in therapy is the examination of the upper respiratory tract for evidence of chronic infection. It is immaterial whether the infection is through the lymphatics, as suggested by Mullin and Fetterolf, or whether the infected material is aspirated during sleep, or both. The fact of the matter is that successful results in treatment depend upon the eradication of the infection located higher up. Often the progress of the bronchitis and the sinus lung, described by Marriott, is dependent upon the success of our nasal treatments.

When I was associated with a sanatorium for tuberculosis we found many lungs supposed to be tuberculous that were sinus lungs. They did well if the sinus disease could be eradicated. Dr. Marriott often sends us cases that have had a diagnosis of chronic pulmonary tuberculosis, with the statement that they are sinus lungs. So far his observations have always been correct. The X-ray picture of a sinus lung is very suggestive of the condition. Marriott[1] states that X-ray of the chest in these cases shows an enlarged hilus shadow and peribronchial thickening, often extending well out into the parenchyma of both lungs. Small dense areas, such as are usually interpreted as shadows of calcified glands, may be seen. Tuberculosis in these cases may be ruled out by negative intradermal tuberculin tests, using increasing amounts of tuberculin. Successful treatment of paranasal sinus infection in these cases results in marked and progressive improvement.

In treating children we must realize that there is such a thing as an allergic nose—that is, one where the rhinitis or sinusitis is the result of a general state allergy. Again conditions about the nose, as sinusitis, may precipitate an allergic attack in an

allergic individual. I do not know if there is such a thing as a bacterial allergy. It is sure that asthmatics cannot be properly treated without taking into consideration the nasal pathology.

The treatment of an allergic nose is allergic. I can see no objection to operating upon an allergic nose in children if they need operating. Many have a suppurative sinusitis in addition to the allergic thing. But to operate for the allergic condition which will disappear with treatment is positively not justifiable. To illustrate: A boy of eleven years had a sinusitis with white discharge coming from both nostrils. The trouble came on in July without history of exposure to inclement weather. He had had the same thing before. Extensive operations on the sinuses had been advised. He had 40 per cent eosinophils in his nasal discharge with only an occasional polynuclear cell. The maxillary sinuses especially were blurred. I insisted that he have only allergic treatment and he completely recovered.

Another case came under my observation. The maxillary sinuses were blurred on X-ray. He had been advised to have a Caldwell-Luc operation but refused. He had no treatment. In a few weeks his sinus plates were clear.

The following case is instructive: The patient, about nine years of age, had a diagnosis of tuberculosis of the lung with asthma. Dr. Marriott diagnosed a sinus lung and transferred the boy to our service. He had a bilateral maxillary sinusitis with no eosinophils in the discharge but many polynuclear leucocytes. For one year he was treated. Washing the maxillary sinuses by needle puncture always relieved his asthma. When ventilation of the sinuses was recommended it was refused and vaccines were used. After two months the father asked me where to take the boy for the winter because of his asthma. My answer was, "Why take him any place when ventilation of the sinuses will give a good result both for his sinus trouble and his asthma?" His asthmatic attacks were always preceded by an acute exacerbation of his upper respiratory infection. Three months ago windows were made in each maxillary sinus. The result is most satisfactory.

We always make nasal smears in examining our sinus cases. Many pus cells in an allergic nose should make us look carefully

for indications for operation in addition to the allergic treatment. The presence of 10 per cent or more of eosinophils without pus cells should make us think that operation was contraindicated and allergic treatment alone be instituted. The presence of pus cells in the nasal or sinus discharge indicates infection. In asthma, even if the case is distinctly sensitive to protein substances other than bacterial products, if sinus suppuration is present it is of great clinical importance and the suppurative process must be treated.

Probably infections of the upper respiratory tract appeal to the pediatrist as being most important, as they relate to encephalitis, rheumatic fever, chorea and nephritis. When we review these cases we are impressed by the fact that when any of these conditions were associated with a severe upper respiratory infection the systemic trouble is influenced tremendously by very slight upper respiratory infections. For instance: A child has a severe upper respiratory infection followed by acute rheumatic fever; with the control of the nose and throat condition the child is much better. Usually the tonsils are removed. The child leaves the hospital in a very satisfactory condition. Later he returns with the history of a "very slight cold," not enough to disturb the average child, and followed by a very bad attack of acute rheumatic fever. The cells of the organism seem to have developed a sensitivity to certain substances produced by the cold.

Through the kindness of Dr. Marriott, I have just reviewed eleven cases of encephalitis preceded by and apparently the result of an infection of the upper respiratory tract. Certainly they all recovered after the disappearance of the nose and throat infections. The age limits of the cases studied are seventeen months and eight years. All of the children were very seriously ill and all but one was given a good prognosis. Apparently one case so simulated tuberculous meningitis that it was only after long study, including the inoculation of guinea pigs with spinal fluid, together with the recovery of the child that the diagnosis was made.

The patients all gave a history of preceding infection of the upper respiratory tract. They had convulsions, coma, paresis with fever. The spinal fluid was under increased pressure, the lympho-

cytes were increased up to 138. With the eradication of the infection about the nose and throat all the symptoms disappeared.

In children chronic heart disease usually occurs as rheumatic heart disease or chronic heart disease without rheumatic fever. There are two other small groups of children: those with congenital heart lesions and those with subacute bacterial endocarditis.

The rheumatic heart disease[2] usually has its beginning in the first ten years of life. Most commonly there are preceding joint pains, but these are not necessarily present. Usually there is a history of repeated sore throats or colds, although the attacks sometimes occur after a first attack of tonsillitis. The focus of infection by far most commonly associated with this condition is chronic tonsillitis; certainly in this class of cases we must assume that as long as diseased tonsils are present the body will continue to react to this infection with unfavorable results. The most favorable time to remove tonsils and adenoids is when convalescence is well established. Many patients will make no progress toward recovery until these and other sources of infection are cleaned out. After the removal of the tonsils these children are frequently free of infection about the nose and throat.

A larger group of children with heart disease[3] are those without joint infection. They complain, however, of obscure pains and stiffness about the joints. There is an unfailing history of infection of the upper respiratory tract. Infection is found in the nasal sinuses or tonsils, most commonly in both. The sinus infection is usually most marked in the maxillary sinuses. There is an intimate relationship between the nose and throat condition. The prognosis of the heart disease is much dependent upon our ability to eradicate the sinus infection. It must be cleaned up properly and kept cleaned up.

Unfortunately it is often quite difficult to get the desired result so far as the sinuses are concerned, and then the heart does not do well. The trouble starts in the nose and the throat, and the success of treatment depends upon the energetic early procedures directed towards infection there.

Rheumatic fever and chorea are preceded by infection of the upper respiratory tract, and the percentage of recurrences are

influenced very much by our ability to combat successfully the infection in the upper respiratory tract.

It is very astonishing how slight infections here precipitate recurrences. A child with chorea or rheumatic fever while in the hospital has the tonsils removed; he leaves in fine condition and returns with the history of a very slight cold, such as would not influence the normal child, precipitating an attack of chorea or rheumatic fever. There seems to be an extreme sensitiveness or allergy to toxins.

Operation for the removal of tonsils in a chorea patient during the quiescent stage may precipitate a dreadful choreaic attack. The pediatric service is trying to obviate this trouble by large doses of salicylates.

While the best time to remove tonsils and adenoids in this class of children is before the disease manifests itself, usually we see the children with the rheumatic fever or chorea. The operative work should be done when the pediatrist deems it best.

The removal of tonsils and adenoids does not prevent chorea or rheumatic fever. It has an influence on its incidence, however.

The relationship between glomerular nephritis and tonsillitis is startling. Blood colored urine preceding the removal of tonsils is often perfectly clear forty-eight hours afterwards. The tonsils are by far the most common harbor for the hemolytic streptococcus. Rarely after the removal of the faucial and pharyngeal tonsils he is in the lingual tonsil and it must come out. Occasionally the organism is in the sinuses or in a discharging ear. Then a satisfactory result will only be secured by the eradication of the infectious process in sinus or ear.

The discussion of the relationship of foci about the upper respiratory tract to Still's disease or chronic arthritis in children must be tempered with conservatism. Certainly these children do better after the eradication of the infection. But any child has its nutrition improved by such procedures. It may be that the improvement is all due to this and that there is no direct action on the disease process. This is a matter that we now have under consideration, and we hope to be able to add something definite in the near future. The clinical course of the patient is so erratic

that it is difficult to evaluate the result of work on the nose and throat.

Malnutrition is a most important problem in infants and children. It is common to observe a child undernourished, languid, underweight and irritable where the best of diet and climatic conditions do not produce a favorable result. In infants, not so often in children, such a condition is often accompanied by gastrointestinal upsets.[4] We all know that removal of infected tonsils and adenoids most commonly cause a wonderful change in the child. The appetite is better, the weight increases, and the child does well in school. It ceases to be irritable; a secondary anemia often present disappears. However, the infection causing the trouble is not always present in the tonsils, or at least it is not limited to the tonsils. At all ages the condition of the nasal accessory sinuses and the ears should be taken into consideration. In every child where the removal of tonsils and adenoids does not give the desired result the ears and the sinuses should be most carefully studied. If infection is found in either its eradication should be of much benefit to the child.

Dr. Jeans,[5] in an address before the American Laryngological Association in 1925, summarized the matter as follows:

Improvement following the treatment of sinusitis in many instances has been so definite and marked as to be convincing as to the role this infection has played. To see a child with deforming arthritis associated with severe bouts of fever become comfortable and afebrile, and to see function gradually restored as a result of treatment of sinus disease is impressive, especially when in former years one has been accustomed to see all efforts unavailing and the disease become progressively worse.

The following conditions in infants and young children may result from a focus of infection or may result from sinus disease: Cardiac lesions, rheumatic fever, chorea, nephritis, pyelitis, certain cases of cyclic vomiting, deforming periarthritis, anemia, anorexia, malnutrition and chronic digestive disturbances.

Remote effects more or less peculiar to sinus disease are bronchiectasis, asthma, infants with cholera infantum symptoms complex.

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LI.

WHAT CLIMATE FOR UPPER RESPIRATORY DISEASES?*

JOHN F. BARNHILL, M. D.,

MIAMI BEACH.

"Go to Arizona and you will be well in a week." This statement was made to a patient who had been operated upon for a chronic frontal sinus suppuration but who continued to suffer severely because of failure of the wound to drain properly or to heal. The patient was in despair and of course eager to journey at once to a land so full of promise. The advice was given by a consulting surgeon in a tone of authority that seemed to admit no argument or doubt. The speaker did not state just where the patient should go in Arizona, nor did he give any directions as to her mode of life after arrival. The inference clearly was that merely to live anywhere in that region for a brief period was all that was necessary. The surgeon had never been in Arizona and knew nothing of its climate or of living conditions there. Considering the pathology present in the affected sinus the advice was absurd. The patient needed a secondary operation and not climate.

The above circumstance is narrated chiefly to call attention to a practice not wholly uncommon, whereby patients are urged to change climate without first having used every means to ascertain the exact extent and nature of the affection from which the patient suffers, or to determine definitely what region is most suitable to the disease in question. One need scarcely say that such loosely given, unwarranted advice can lead but to grief of all concerned, to financial hardship in many instances, and of course ultimately to loss of confidence in medical leadership. Over-enthusiasm on the part of some physicians in recommending climate as a positive cureall no doubt originated to some extent in regions most reputed to possess the valuable asset. Local

*Presented before the American Laryngological Association, May, 1930.

pride and commercial interests indigenous to favored climatic areas make difficult the path of one who wishes to arrive at exact, practical facts. Boosters' and optimists' clubs, realtors' boards, Chambers of Commerce, publications of many kinds and even railroads have fathered glowing accounts of their respective resort countries. These have had much influence in shaping the views of both physicians and laity concerning the value to health from residence there. Careful observers ever since the time of Hippocrates have believed in the value of climate as a therapeutic measure. It is reasonable to assume that a view held for so many centuries is still worthy of sincere consideration. Therefore the criticism just offered is directed against unjustified enthusiasm, and is not intended to discourage the employment of a valuable medical asset.

The belief so tenaciously held by the laity and a large portion of the medical profession that the southwestern and southern portions of our country possess more health giving, health restoring virtues than the more rigorous regions to the north have been controverted lately but not shaken, by geographers and other scientific workers, including a few physicians. The geographers claim to have proven that the climate of the more northerly region is better than that lying to the south, and certain physicians have concluded that in the matter of taking cold, an important item in the inception of many serious diseases, the question of climate is of little or no importance. Since this conclusion is quite revolutionary, it is, perhaps, well to state the bare facts on which it is based. I shall confine the summary to the investigation of W. H. Barrows, of Stanford University, whose work and deductions are very similar to others. Barrows' researches related to the question of the relative frequency of colds among students in widely separated universities and under vastly different conditions of climatic environment. At Stanford where the climate is mild, equable and indeed rather ideal, practically the same number of men per hundred among those investigated suffered from coryza under every circumstance as to mode of living, as suffered from the same affection among students at Harvard, which is located in a comparatively rigorous climate. Likewise women students of Stanford, and women

students of Wellesley, the latter also in a cold, comparatively disagreeable climate, living under similar conditions in every particular except climate, also were affected in each institution by practically the same number of infections of the upper air tract. Barrows drew the conclusion from these studies, as did all the others who pursued similar investigations, that climate is not a major factor either in the production or avoidance of colds.

The conclusions of geographers, although conducted along entirely different lines from those of Barrows and others, is equally interesting and, one may add, rather surprising. Huntington, a professor at Yale, discusses among other questions relating to climate and civilization, the important item of the ideal climate, and leads the reader to infer that the most healthful parts of the United States center about New England, the St. Lawrence river country, and the region around the Great Lakes. Huntington's conclusions are based not alone upon his own extensive researches but also upon the researches of many other presumably noted geographers. Among other interesting things concerning the ideal climate, Huntington states that the average annual temperature should be near 64° F.; that neither extreme heat or extreme cold is ideal, but that regions having a considerable difference between day and night temperatures should be regarded as favorable conditions. He further argues that sections having frequent, violent storms are most suitable places of residence as regards health; that an atmosphere well saturated with moisture is better than a dry one, and that but a moderate number of days of sunshine is preferable to a constant or almost constant succession of days having clear skies. It must be evident from a study of the work of this author that he does not regard those regions of the United States which are commonly regarded as having a climate of superior quality, as any better, if as good as those parts which are known to be cold, damp, changeable and altogether wholly disagreeable during the winter season. In any attempt, therefore, to estimate the value of climate in relation to disease the physician must form his own estimate as to which of the foregoing opposed opinions is most nearly correct.

This brief paper is an effort to analyze existing differences of opinion and to arrive, if possible, at some conclusion which may be used as a safe guide in practice. To do this I have consulted in addition to the research work already quoted, the records of the United States Weather Bureau; reports of insurance companies, a few of which have lately given considerable intelligent effort to ascertain the cause of infections of the upper air tract; otolaryngologists who are known to be competent observers and reliable reporters; and lastly I have turned to whatever profit I might, my own observations, gained by extensive travel and numerous, comparatively brief periods of residence in favored resort places. I fully realize that my own personal observance covers periods entirely too brief to be of great value except in the matter of estimating the worth of other information.

I regret that some of what I have to say seems personal and dogmatic, but this treatment of the subject appears necessary since our present knowledge is limited, and therefore all discussion must be more or less doctrinal. I trust such errors as are made will be regarded as errors of observation and will not be looked upon as expressions of bias or prejudice.

Statistical information, as well as my own tabulation of facts leads to the conclusion that there is no perfect climate anywhere. Many boosters say there is, but even the greatest enthusiasts of the most favored regions privately admit slight defects in their own environment. These deficiencies vary in different regions: in one that may be widely heralded as perfect in every respect it is admitted that there is too much rain or fog; or the difference between the temperature of midday and midnight is too great. In another it is too dry, and there may be too much sunshine. In still another, perfection of climate is marred only by an occasional drop in temperature, of from 15° to 25°, which is usually accompanied by disagreeable winds, and lasts three or four days. Then, too, in some otherwise splendid climatic areas of the desert, occasional dust storms occur which lower the climatic rating.

All the above enumerated imperfections are but trivialities to those in good health, and they may mean little even to those who

suffer from changeable temperatures, provided proper clothing, shelter and heat are provided. Mention is made of them for the reason that suffering and ultimate harm is almost certain to come to those who seek such climate in the belief that no bad features are ever met with, and consequently go unprepared to meet them.

The crowded waiting rooms and busy lives of physicians who practice in health resort places seem to belie the reputation of such regions as health giving localities, and to indicate that the climate is neither a preventive of disease nor a cure for any affection. While physicians admit that infections of the upper air tract are frequently met with in their favored climatic environment, they are quick to explain the circumstance on the ground that such places are filled with visitors from less favored regions and that these bring with them the foci of infection from which they formerly suffered. They seem certain that the course of such infection is not so severe, and not so prolonged as in less favored climatic regions. All stoutly maintain that complications occur with less frequency and that sequelæ are less common than elsewhere. They are keen to assert that those who take cold readily in the good climate, do so not because of infection from without, but rather from autogenous sources which the patients carried with them from a former environment. These arguments are, I believe, substantially correct, for it is undoubtedly a fact that a large percentage of all disease treated in resort places is imported: and in the case of upper respiratory infections it is a fact, usually demonstrable, that persons who harbor chronic foci of infection often reinfect themselves, producing thereby all the symptoms of a primary acute rhinitis or other inflammatory ailment. It is just this type of patient, the one who carries a chronic focus, who is seldom free from cold in a rigorous climate, that most readily suffers a breakdown, even in the most favorable climate on the occasion of sudden temperature changes. This brings back again the question of what constitutes a favorable climate. Clinical experience is not in accord with Huntington that the most favorable climate is one in which the difference in temperature between day and night is quite marked. Indeed clini-

cal experience convinces one that the exact opposite is true, and that but slight changes during the twenty-four hours have great advantages in the climatic therapeutics of a large number of infectious conditions of the upper air tract.

A mild, equable climate, one having no marked or sudden disturbances of temperature, is best because it is most suitable for living in the open. The benefits to be derived from outdoor life are easily understood if it is recalled that a majority of all diseases of the respiratory apparatus are of bacterial origin, and that such affections are spread from one person to another, chiefly in confined, stuffy places. This fact is exemplified during the winter season in every region of the north. Confinement indoors and close contact with infectious individuals is common, with the result that many suffer more or less constantly from October to May from some form of respiratory affection. Should these same persons change residence to some salubrious region of the south or southwest, where outdoor life is almost constantly invited and practiced, most former infections will disappear. I have personally witnessed at one playground in south Florida a striking example of this fact. The place is called the old man's paradise. It would seem well named, for one may see on any day during the winter season thousands of aged men and women at play in the open, all wildly gesticulating, talking loudly, or even quarreling violently just as children do at sport. All are having a happy time of it and few are ever heard to cough or sneeze.

While witnessing an outdoor exhibition such as this in midwinter, in which so large a number of elderly people are joyously participating, the observant physician inevitably recalls his clinical experience with old people in a rigorous climate and honestly concludes that were these same men and women in their northern home, practically all would be confined in stuffy, heated rooms; many would be suffering greatly from respiratory affections, and not a few would be seriously or even fatally ill from infection, or some sequel of infection.

What I have just described at the old man's paradise I have seen enacted differently under different circumstances and environmental conditions from Los Angeles across the continent

to Miami Beach, for the climate of many favored places is such as to encourage life in the open with comfort, and the effect on those who avail themselves of the advantage is much the same everywhere. An enthusiast once stated to me as a fact that when old people live as long as they can in a bad climate, should they then come to his most favored climate, they always live ten years longer. There is, one must admit, some basis of truth in this assertion.

The preceding discussion of this subject has little value unless from it some practical deductions may be drawn. Two questions have chief interest: What class of patients do best in the most favored climates, and which among such favored regions is best for the individual patient? These questions can be answered only in a very broad and general way. First, as to what type patient? The class of patient most benefited is that characterized by the person whose resistance is low, who takes cold easily and who suffers more or less constantly from some form of inflammation of the upper air tract from the first frost in autumn to the last frost in spring. This group includes moderately mild cases of paranasal sinus infection, especially of the ethmoids, rhinitis, pharyngitis, bronchitis, and asthma due to bronchial inflammation. The type embraces a large percentage of all diseases affecting the upper respiratory tract. Mild, though intractable suppurative processes of the middle ear and mastoid process often promptly improve, or heal completely in a favorable climatic environment. However, it is neither justifiable or expedient to send a large percentage of such patients away from home environment. Change of climate is in most instances still a rich man's experiment, and therefore to send anyone away unless that person has ample resources, or is fully assured beforehand of means of comfortable existence in the new environment, is to approach dangerously near to the commission of an inexcusable wrong.

Types which are but little benefited, or are not at all improved by climate, include accessory nasal sinus suppuration in which the affected sinuses have suffered marked pathologic change either of mucosa or osseous structure; also affections of the middle ear in which areas of necrotic bone, or diseased and

decaying ossicles are present, and mastoiditis, the chief pathology of which is cholesteatoma, sequestra, or bony necrosis. No stage of syphilis of the nose, throat, or ear is directly benefited, and of course affections such as otosclerosis are not improved, except through mental and physical betterment which sometimes are helped as are other bodily ailments. Another class but little if any benefited, is that of nasal deformity, either congenital or acquired, but which results in more or less complete obstruction to respiration. It is, of course, irrational to advise patients such as these to change climate in the hope of receiving permanent benefit. Seriously affected sinuses, chronically enlarged and infected adenoids and tonsils, nasal deformities, from whatever cause, and mastoiditis of a nature above described, should be operated upon in any climate. After operative measures have been carried out successfully residence in a favorable climate is undoubtedly beneficial.

Second, how may the physician select the most suitable climate for the individual patient? This question is difficult to answer satisfactorily, for the reason that our most favored regions have not been well and scientifically mapped and charted from the standpoints of health and sanitation, and consequently much of the knowledge most readily available is little more than private opinion. L. I. Dulin, statistician for the Metropolitan Life Insurance Company, comments, as have others, on our tardiness in capitalizing and utilizing our valuable climatic assets. Dulin says: "The Committee on Public Health Climatology of the American Public Health Association has been trying over the past few years to secure some support for the preparation of an atlas of medical or public health climatology for the United States similar to Europe. In Switzerland, physicians also have been most active for nearly half a century in collecting physiologic data in association with climatic data. We have no effort of that kind under way in the United States at the present time."

Fortunately the wide extent of our southern and southwestern country provides a great variety of choice in the selection of a region having a climate suitable to the comfort of the patient and the needs of almost any disease. Including a broad belt from ocean to ocean as it does, and crossing mountain ranges, plateaus,

deserts and fertile plains, many environments may be found that are cold in elevated places, subtropical near sea level in the extreme south, hot and dry with perpetual sunshine in desert stretches, and cool and pleasant with considerable fog in certain coast districts.

Most patients who suffer from infection of the upper air tract and from bronchial inflammation do best in a region that is moderately dry, has an abundant but not perpetual sunshine and one that is not characterized by marked and sudden temperature changes. In brief a region which provides a climate that makes continuous life out of doors possible, comfortable and pleasurable. It should, of course, be understood that the most nearly ideal climate is usually valueless until modern sanitation, including provision for pure water, correct housing, proper drainage and the elimination of insect pests, has been added. The most favorable climate of any region must forever be linked with the advances of civilization and science.

There are numerous regions in our southwestern and southern states which closely approach the conditions of most favorable climate. All are, of course, not equally good in every respect, but many well known resort places in Arizona, New Mexico, and Texas have praiseworthy climates to which properly selected cases may be sent with assurance of benefit. However, sections which most nearly attain the ideal are the coastal regions of southern California and southern Florida. And, assuming that the item much emphasized in this paper is correct, namely, that a close approach to equality of temperature of day and night is best for the type of affections under consideration, then, of these two the southeastern coast of the latter from Palm Beach to Key West has some advantage because of close proximity of the Gulf Stream. The nearness of the great natural, warm water current assures the neighboring shoreland of a climate almost constantly suitable to outdoor life, and hence is well adapted to the needs of those who suffer from certain well known types of respiratory disease of the upper air tract.

There is substantial basis for the assertion that in portions of our southernmost states there are climatic conditions that are equal, and in very many instances quite superior to the much

vaunted resorts in foreign lands. And, while it is true that much of this vast stretch is not developed, and is not at present suited to the reception of ailing visitors, there are numerous regions and many institutions already established that are giving commendable, highly satisfactory service to those more seriously sick. There are also climatic environments where, chiefly through living in the open, those less seriously infected may regain health in a wholly pleasant and agreeable way.

Because of the very limited time for presentation of this paper it has been impossible to present the subject with any degree of fullness. On this account I wish now in closing to emphasize the following points:

1. There is regrettable laxity on the part of some members of the profession in prescribing climate.

2. There is no perfect climate. Occasional unpleasant conditions arise, even in the best, which may cause discomfort with backsets, and even danger to life, unless anticipated and sensibly met.

3. The best climate is unfavorable unless a modern sanitary environment has also been established.

4. The most valued features of any climate, in so far as they relate to benefit of respiratory infections, are mildness and freedom from marked change of temperature from midday to midnight.

5. Respiratory diseases most benefited are those infections the pathology of which is not greatly advanced and emphasized.

6. That climate has best therapeutic value in the relief of respiratory infections which permits a maximum of pleasurable, comfortable outdoor life.

LII.

ADENOMA OF BRONCHUS.*

RUDOLPH KRAMER, M. D.,

NEW YORK.

Adenomata of the bronchus are apparently a rarity in medical literature inasmuch as I have been able to find only five cases on record. However, it is not because of this infrequency of occurrence that this report is being made, but because of the clinical course, prognosis and treatment of this class of tumors. I believe that adenomata of the bronchus are not as rare as the literature would lead us to conclude, but that with increased use of the bronchoscope in the diagnosis of pulmonary lesions of all varieties and with a better understanding of the microscopic pathology and the clinical course, these growths will be found more often. Even today, too many cases of pulmonary disease are allowed to go without bronchoscopic examination, and as the clinical picture resulting from adenoma of the bronchus is that of its secondary effects, thus simulating many different conditions of the pulmonary field, it follows that the presence of this bronchial growth will be overlooked. Furthermore, the amount of tissue endoscopically removed, when this is done, may be relatively small, and, as the microscopic diagnosis is often exceedingly difficult, an erroneous diagnosis is made of carcinoma or endothelioma. The clue to the correct diagnosis in these cases was in a postmortem examination and a minute study of a polypoid growth in which the microscopic picture was established. The entire clinical and pathologic study of adenoma of the bronchus is taken up by Dr. Harry Wessler and Dr. Coleman Rabin in a paper which will appear shortly in print, on "Benign Tumors of the Bronchus," in which paper they report eight cases of adenoma. The diagnosis of adenoma having been made, the prognosis and treatment fall in definite lines. The prognosis, instead of the fatal one of

*Read before the thirteenth annual meeting of the American Bronchoscopic Society, at Atlantic City, N. J., May 27, 1930.

an erroneously diagnosed carcinoma, becomes a relatively favorable one. The treatment is either by endoscopic surgical removal if this is feasible, by radium implantation, by coagulation (a method which I have not employed), or a combination of these methods. Inasmuch as this lesion will be thoroughly considered in the above mentioned paper of Drs. Wessler and Rabin, I shall omit any discussion of this class of tumor and recite the history of two cases which have been subjected to treatment for some time and conclude with a statement of the pathology of these cases by Dr. Rabin from the above mentioned paper.

Case 1.—Female, 36 years of age, referred by Dr. A. Jaffin of Jersey City, in February, 1929, because of hemoptysis for one year at about monthly intervals, generally coinciding with the menses. The quantity of blood raised at any one time was as much as three ounces. There was, in addition, sticking pain in the right chest on deep inspiration. There was a history of pleurisy with effusion thirteen years before, lasting three months, and frequent attacks of the "grippe" for nine years.

On physical examination there was dullness, diminished breath, voice and whisper over the right lower chest, absent fremitus. Diagnosis on admission, tuberculosis or neoplasm. X-ray examination showed an infiltration, pneumonic in type, probably in the right middle lobe. Pleura thickened at right base. Diagnosis, pneumonia or carcinoma. Dr. Wessler made the diagnosis of benign neoplasm rather than malignant neoplasm of right middle and lower lobe bronchi for the following reasons:

1. History of menstrual hemoptysis, not associated with cough or expectoration.
2. Excellent condition of patient one year after onset of symptoms.
3. Evidence of bronchial stenosis.
4. Absence of clubbed fingers.

Bronchoscopy under local anesthesia showed a rounded mass covered with normal appearing mucosa occluding the entire lumen of the right bronchus just below the level of the middle lobe orifice. A specimen was removed for microscopic examination. Bronchoscopic diagnosis, malignant growth of bronchus. Pathologist's diagnosis of tissue removed, adenoma of bronchus.

Bronchoscopy was performed every two weeks and tissue was removed but growth maintained its size. On May 4, 1929, three removable radium seeds were inserted into the growth and were removed six days later; a total dosage of 700 mc. hours. On June 20th, a note was made that the growth was smaller and three removable radium seeds were again inserted. These seeds were removed seven days later—the total dosage was 800 mc. hours. Again on December 20, 1929, three seeds were inserted and removed six days later. Since that time the patient has ceased bleeding, the mass has become smaller, although it still occludes the middle lobe branch and part of the lower lobe branch. Repeated microscopic examination of specimens removed at intervals from the growth show granulation tissue and no evidence of adenoma. (Dr. Klemperer, pathologist to Mt. Sinai Hospital.) The patient is still under treatment; she still has some cough and at present we are contenting ourselves with removal of the presenting growth. We propose to use electric coagulation on the remaining mass if surgical removal does not yield satisfactory results.*

Case 2.—Male, 26 years old, seen March 11, 1926. He was complaining of cough of three years' duration after pneumonia. He had pain in the left lower chest posteriorly which was diagnosed as pleurisy. The chest had been tapped with negative findings, after which the pain cleared up. He had lost 25 pounds, felt weak, and was advised to go to Denver, Colo. Later he had cough after slight exertion and on arising. This cough occurred in spasms; there was no expectoration and no pain. Eight months before admission he expectorated a teaspoonful of blood. This cough occurred in spasms; there was no expectoration and no pain. Eight months before admission he expectorated a teaspoonful of blood. This occurred twice in Colorado. X-ray of the chest was negative two years before we saw the patient. His cough became worse and he became dyspneic on exertion. Sputum examination for tubercle bacilli was negative. On physical exami-

*Since this paper was read, on May 27, 1930, electrocoagulation of the growth through the bronchoscope has been done with marked recession in the size of the growth.

nation there was dullness on the left side below the sixth rib, slightly diminished breath sounds, sonorous rales, absent fremitus, faint voice and breath sounds. Provisional diagnosis, unresolved pneumonia with chronic pleurisy of left base. X-ray examination showed a dense shadow in the left lower lobe, left leaf of diaphragm elevated. Diagnosis, possible neoplasm. On September 2, 1926, bronchoscopy was performed under local anesthesia. A fungating mass which completely obstructed the left lower lobe was found and a specimen removed which was at that time diagnosed as carcinoma. The patient was lost sight of until February 17, 1928, when he returned complaining of slight fever and purulent expectoration. There were signs of obstruction of the entire left lung now. Bronchoscopy under local anesthesia showed a pinkish lobulated mass arising from the left main bronchus on the mesial wall just above the level of the upper lobe branch. A large portion of the growth was removed with forceps and snare. Diagnosis, adenoma. On March 23, 1928, bronchoscopy was again performed under local anesthesia, and on the mesial wall of the left main bronchus opposite the left upper lobe branch there was a mass 1 cm. in length, 7 mm. in width, reddish in color, covered with apparently normal mucous membrane. From the anterior portion of this mass there was a thin edge of apparently normal mucosa running across the anterior wall of the bronchus to the lower lip of the left upper lobe branch. The lower lobe bronchus was dilated and contained thick secretion. Three removable radium seeds were inserted into the mass parallel to the course of the bronchus. These seeds were 4 mc. each. Five days later they were removed. On May 17, 1928, bronchoscopy showed no evidence of any growth. There was a scar on the median wall of the left main bronchus. On June 27, 1929, bronchoscopy showed a depressed scar about 3 mm. in depth on the mesial wall of the left main bronchus about 2½ cm. below the spur. The patient was perfectly well and has remained so.

The removable seeds are introduced by means of Dr. Sidney Yankauer's introducer which I shall be glad to show you.

Because of the importance of a proper understanding of the pathology of these tumors, in order to adequately treat them, I am including the following report by Dr. Coleman Rabin.

In addition to the fibromas, the fibrolipomas and the enchondromas of the bronchus, there is a group of adenomatous polypoid tumors which in general are characterized by a prolonged clinical course. The cause of death in these cases is infection of the lung distal to the obstructing endobronchial tumor. Eight cases of this type have come under our observation. In three, autopsies were performed. There were no metastases in any of these cases. Three of the cases are still under observation. The other two have disappeared from our observation.

These neoplasms are almost always smooth, round or oval, polypoid tumors arising from the main bronchi or from the bronchi of the first order. They may be pedunculated or may arise from a rather wide base. It is quite characteristic for them to project towards the trachea; those arising from the main or lower lobe bronchus pointing upwards, and those from the middle and upper lobe bronchi peeping into the main bronchus. The reason for this peculiarity of growth is quite obvious. The expulsive effort of coughing forces the polypoid growths outwards. It is to this that we owe the ease of recognition of the tumor under the bronchoscope. To this peculiarity, also, we must attribute the fact that a polypoid tumor of a branch bronchus may affect a large portion of the lung.

The surface of the tumor is characteristically smooth; the more exposed portions of the bronchial epithelium which covers the tumor often show a remarkable degree of squamous metaplasia, resulting in the formation of hornified, stratified, squamous epithelium. In the more protected basal portion the bronchial epithelium may appear normal or may be made up of a stratified layer of columnar epithelium. Beneath the epithelium there is a completely intact, well developed basement membrane which intimately covers the subjacent tumor tissue. This description applies to the cases which have come under our observation and to the cases reported in the literature.

The tumor is made up of rather small cells, the cytoplasm of which varies in size in different cases. The nuclei are usually small. They are either perfectly round or inclined to an oval shape. The striking characteristic, however, is their uniformity and the homogeneity of their structure and staining qualities.

They are usually arranged in acini with perfectly definite basement membranes. However, frequently this acinar structure, if present is not obvious, and the cells appear to lie in solid masses. There is a tendency of retraction of the cells from the basement membranes in the process of fixation and embedding, which is more marked perhaps than is seen in most adenomatous tumors.

A study of the literature discloses a few cases of this type which the authors consider to be benign. A number of such tumors, however, have been considered to be malignant in spite of the fact that the tumors occurred in comparatively youthful persons, the clinical course was prolonged, and no metastases were found at the postmortem examination.

In the descriptions of the latter, the authors frequently mention that there is an infiltration of the submucosa between the cartilages and in some instances extending behind the cartilages. This, however, does not signify true infiltration. Study of the anatomic distribution of the bronchial glands shows them to be normally situated in exactly these locations. They lie not only on the internal surface of the cartilages but also between and behind them. The presence of neoplastic tissue in these locations, therefore, does not signify infilration, but rather indicates the point of development of the tumor. The significance of this location is a clinical one rather than a pathologic one. It makes for greater difficulty of removal.

In two of the cases which we have observed at postmortem examination there was more definite evidence of infiltration. There were occasional groups of cells which apparently lay in the stroma outside the glandular acini. In spite of the prolonged history of pulmonary disease and the lack of gross infiltration or metastases at the postmortem examination, such cases cannot be considered as simple adenomas in a pathologic sense. They must be relegated to the group of carcinoma. Whether they eventually would have displayed their malignant character by causing gross malignant changes remains a matter of conjecture. That this might take place is indicated by two other cases which have come under our observation, one with a history of six years' duration and the other of nine years' duration, both of which in the latter

part of their course showed conclusive evidence of malignancy. A similar finding is reported by Heck.

The adenomatous polypoid tumors consisting of small round uniform cells arranged in acini and covered with a smooth lining of bronchial epithelium, whether or not they show minute evidences of stroma infiltration on microscopic examination, constitute a distinct group. They have no counterpart in the ordinary form of bronchial carcinoma. The latter characteristically run a very rapid downhill course with extensive metastases. The former run a prolonged course, a rare finding in the ordinary types of bronchial carcinoma. Those cases which show no definite infiltration of the stroma are justifiably considered as pure adenomas. Those which show infiltration of the stroma are pathologically carcinomas. Clinically, however, there is at present no justification for the belief that the latter invariably will give rise to gross infiltration of the lung or metastases. There is no doubt that some of them, however, will eventually manifest their malignant nature by running such a course.

The point of origin of these tumors appears to be quite definite. That they do not originate from the bronchial epithelium is shown by the fact that the tumor is invariably covered by this epithelium which is separated from the tumor by a complete basement membrane. The cells do not resemble those of the mucous glands. They are smaller and the nuclei stain more deeply. We have observed the mucous glands to be distinctly separated from the tumor with no apparent transition. The cells composing the tumor very closely resemble the cells of the ducts of the mucous glands. In one of the cases there was conclusive evidence of proliferation of the ducts. In parts of the tumor dilated ducts were found which had undergone squamous metaplasia, so that some of the acini were composed of stratified squamous epithelium. In the case reported by Horn the tumor was composed of acini lined by ciliated epithelium. From these observations we must conclude that the point of origin lies in the ducts of the mucous glands.

LIII.

THE PREVENTION OF NASAL DEFORMITIES.

WILLIAM WESLEY CARTER, A. M., M. D.,

NEW YORK.

Preventive medicine has, for so long a time, monopolized the stage that procedures designed chiefly to anticipate and render unnecessary future surgical operations may not attract the attention they deserve. I know of no part of the body where skilled attention at the time of the injury yields more brilliant results than in the nose.

The nose is not what might be correctly called a vital organ, for one might exist without one, and yet I fancy that few people could be found who would consider life worth while if a hole in the face were substituted for this very serviceable and conspicuous organ.

From a physiologic point of view, the chief functions of the nose are as the organ of sense of smell and as a respiratory passage where the inspired air is warmed and moistened before entering the lungs. Perfect health is incompatible with the loss of either of these functions. A third, and no less important function, is one chiefly of esthetic value, its shape, size and relation to its facial environment.

To a large extent the development of a normal nose, both from a functional and esthetic point of view, is due to the care which this organ receives during childhood and early adult life. The growth of the immature, flattened nose of the infant into the organ of adult proportions is to a marked degree a mechanical proposition, and a perfect result is dependent upon the correct alignment of the mechanical forces that are involved in its construction and the preservation of this alignment until nasal development is complete, which does not occur until general maturity has been established. There is an old saying, "A baby lives half of its life on its nose." This statement savors of flippancy

and, of course, cannot be accepted literally, but we know that few children grow up without having had more or less serious injuries to the nose. I feel quite sure that these traumatic injuries in early life which were not regarded at the time as of any consequence and which were treated with a few kind words, cold water to stop the bleeding and in some instances a strip of adhesive plaster across the nose, may account for the fact that nasal septa, in 75 per cent of adults, are deflected and that a perfectly symmetrical adult nose is of sufficient rarity to be highly prized by its possessor. I realize that the truth of this suggestion is difficult to prove by clinical observation, as the deformity does not occur coincidentally with the injury; the development of both the intranasal and external deformity is gradual and proceeds pari passu with the growth of the child. Therefore the process by which the nose becomes obstructed and deformed in later life can be understood only by making a careful study of the dynamics of normal nasal development. We are then able to predict what the result will be when these forces are perverted by traumatic interference with their action.

I will not enter into a discussion of the embryology of the various structures of the nose, as this is intended to be a practical presentation of a subject that has been woefully neglected.

The development of the nose may be considered as similar to the process of raising a tent, the skin, nasal bones and soft parts of the nose corresponding to the canvas and the septum and nasal processes of the superior maxillæ to the tent poles. These supports are under continuous vertical pressure from early childhood, and if, during this period of development, they are thrown out of line and are unable to regain their normal plane of action, a deformity, usually attended by nasal obstruction, is certain to follow, either at the time or later in life, owing to imbalance in these constructive forces.

During babyhood, when the tissues are resilient and the elevating function of the septum and nasal processes of the superior maxillæ have not yet become active and consequently they are not under vertical pressure, it is easy to conceive that the nose receives many severe blows from which it recovers spontaneously

and without liability to future trouble, but after development is under way and the above mentioned structures are under vertical pressure and have acquired a certain amount of rigidity, the conditions are entirely different. At this time, if the septum, through which the chief elevating force is transmitted, happens to be thrown out of the vertical it may not be able to spring back to its proper place again. The pressure continuing, as growth proceeds, the curve in the septum becomes more pronounced and we have obstruction and eventually a twisted or otherwise asymmetrical nose.

In my study of a large number of skulls in Bellevue Hospital, I was forced to conclude that the septum is of paramount importance in the development of the nose. In my private and hospital practice, among the large number of patients examined, I do not recall ever having seen a markedly deflected septum in an otherwise perfectly symmetrical nose. I regard developmental asymmetry as inseparably associated with a deflected or otherwise abnormal septum.

Now the question arises, How are we to anticipate and prevent deformities and nasal obstructions that become clinically apparent only after the period of growth is well advanced or completed? This question can be easily answered, but I am skeptical as to whether the remedy can be extensively applied, for, of course, the average layman is uninformed in regard to the dynamics of nasal development, and consequently he looks upon an injury to his child's nose as a matter of little consequence, so the rhinologist is often not consulted until after obstruction and deformity are established and the only recourse is an operation.

There is only one way in which this important situation can be met and dealt with satisfactorily, and that is for the child to be placed in the hands of a competent rhinologist as soon after the injury as possible.

The diagnosis of fractures of the nose is more difficult in the child than in the adult; a positive conclusion, in some cases, can be reached only after most careful examination, it may be under a general anesthetic. Little can be gained by inspection, unless the patient is seen immediately after the injury, for the swelling

masks the extent of the injury, and we may have to wait until this has subsided.

Palpation also may be of little service on account of emphysema or blood crepitus, both of which closely simulate bone crepitus. Here, too, doubt may be dispelled by waiting until the swelling has subsided. The delay, however, should not be extended too long; we should reach a conclusion as soon as possible, for healing in this locality occurs more quickly than in any other part of the body, and it is important that accurate adjustment of the fractured parts should be secured before fixation is established. In injuries to the nose, bony fixation occurs, even in the adult, in twenty days after the accident, so it is plain that no time is to be lost in setting the fracture. When callus has formed with the parts in a false position it is more difficult to secure a good result and the operation may be more extensive than a simple adjustment of the segments.

The X-ray, as a means of detecting a fracture of the child's nose, is of little value. This objection, however, does not apply to adults, where the bones throw more distinct shadows and the conditions for securing a satisfactory plate are more promising.

TREATMENT.

I have found that it is better to wait until the swelling subsides before attempting to adjust the segments of a broken nose. If, however, the patient is seen immediately after the accident, and if there is only moderate subcutaneous hemorrhage and no emphysema, it may be possible to line up the parts in their proper position, but if this is done no external splint should be applied, lest forcible restriction of the impending swelling should cause necrosis of the soft parts; this would be particularly apt to occur if the wound happened to be infected.

Strict cleanliness and accepted aseptic precautions should, of course, be observed. It is better to irrigate the nose at once with an alkalin solution. External wounds should be painted with iodin, followed by alcohol and then closed with dermal sutures: I have found Bauer & Black 000 very satisfactory for this purpose.

After swelling has subsided sufficiently to enable us to make an accurate estimate of the damage done to the structural framework and we find that the nose has been fractured, the parts should be carefully lined up by means of my moulding forceps (small size). One of my gold wire splints should then be placed in each nasal cavity and the entire nose should be immobilized and protected by one of my tin lined copper splints, which should be well padded with gauze and held in position by means of adhesive strips.

Both the intranasal splints and the external splint should be worn for about a week. The nose should be inspected and cleansed each day.

Following an injury of this kind, the patient should be seen by the surgeon from time to time over a period of several months, in order to make sure that the parts are in proper position and that the developmental forces are being directed along normal lines.

Unfortunately, idealistic suggestions, especially if they happen not to deal with vital factors, seldom meet with popular support, and unless one's suggestions are attended by clinical demonstrations even the profession is inclined to turn a deaf ear. I feel, however, that no one who carefully reviews his personal clinical experiences in rhinologic problems will pass lightly over the argument I have tried to set forth in favor of close attention being given to minor injuries to the nose during early childhood, with a view to limiting the number of obstructed and deformed noses seen later on in life.

2 East 54th Street.

LIV.

OSTEOPLASTIC RESECTION OF BOTH THE ALVEOLAR PROCESSES ACCORDING TO KOCHER.

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Tomsk, Siberia.

The removal of nasopharyngeal tumors is one of the difficult problems of surgery. Inaccessibility, profuse bleeding at the time of removal and the need of operating radically have for a long time compelled surgeons to look for a shorter and freer access which would secure a large operative field as well as rapid control of bleeding and prompt removal of the tumor.

To precede the removal of the tumor, a series of preliminary operations have been elaborated. Special osteoplastic procedures —that is, temporary resections of the facial skeleton—have been worked out.

All of these preliminary operations have been designed to secure the more ready access to the tumor, either externally, with the temporary resection of the bones, or the complete resection of the whole of the upper jaw;[1] or through the oral cavity with partial or complete osteoplastic resection of the bones of the hard palate.[2]

In spite of the number of proposed preliminary operations, it has been impossible, up to the present time, to establish a classical method which would meet all requirements. I am inclined to believe that this will be impossible, for each tumor must be considered on its own merits. Nevertheless, each of them, however minutely detailed and well grounded it may be, has defects of its own. To these, in the preliminary operations, should be referred the indelible traces, from the faint scar to the complete disfiguration of the face, resulting from partial or complete resection of the upper jaw.

In operations on oral, palatal and alveolar regions, the above defects do not appear, but these operations, especially the alveolar

ones, are usually complicated by profuse bleeding during the resection of the bones, which arrests the normal progress of the operation. The cosmetic effects of the preliminary operations are unquestionably in favor of those performed through the oral cavity.

Certainly the natural way—that is, the oral cavity without any preliminary operation—should be considered as the best approach to nasopharyngeal tumors. Unfortunately, this is far from true at present.

In my surgical experience, I have had thirty cases of various tumors of the nasopharyngeal tract, twenty-five of them being those of fibromas of the nasopharynx; the rest were malignant tumors.

When operating upon fibromas, in seven cases, I performed the preliminary operations according to Langenbeck, Weber, Ollier, while in all other cases the operations were performed under local anesthesia by means of splitting and slicing off the nasopharyngeal tumor and cutting off the pedicle with a long blunt-edged knife introduced through the nasal cavity under the control of a finger of the left hand through the mouth.

In many cases of the latter group fibromas were as large as a mandarin, and the preliminary operations were forced upon one, but after a certain experience I could readily dispense with them, the fibromas being removed successfully and without any relapse.

In five cases of nasopharyngeal tumors, accompanied by their ingrowth into the neighboring cavities, I have had to resort to the Kocher alveolar method as giving a wider approach to the operative field and causing no disfigurement of the patient's face. Of these, two were cases of nasopharyngeal fibroma and two of sarcoma. In one case a Kocher operation was performed twice due to the recurrence of the fibroma.

Case 1.—P. B., male, aged 65, machinist, was admitted to the clinic January 14, 1925, complaining of nasopharyngeal tumor and reduction of hearing on the right side. Otherwise he had been in good health all his life; married; had had fourteen children, of whom six are living.

In the last few years he has had three attacks of rheumatism. About a year ago his right ear became obstructed; there was a purulent discharge; the left ear had become stuffed intermittently; it has been gradually so with the nose. Three months before admission the nose was entirely blocked.

The general condition was weak; general anemia; muscles withered; he breathed through the mouth. The anterior rhinoscopy showed: Right, deviation septi nasi; in the posterior half of the nasal cavity a tumor was seen filling up its lumen; left, the choanal region was filled up with the tumor. On the right, the tumor had grown into the sinus maxillaris; same on the left. There was a row of carious teeth. Tonsils small, with lacunæ. In the nasopharynx the tumor was seen, which filled the whole of the nasopharynx and dislodged the soft palate downwards and forwards.

The tumor was as large as a mandarin, of irregular rounded shape, of solid consistence; bled when touched. In the right ear, otitis media perforativa purulenta; in the left ear, otitis catarrhalis chronica. Diagnosis, sarcoma.

On January 29, 1926, a Kocher operation under local anesthesia was performed, and the following was found: On the right the tumor grew into the sinus maxillaris; same on the left. The tumor was removed. The patient stood the operation well. Upon the tenth day the patient ate some food; the alveolar processes stood in the right manner; there was a slight mobility in them; the morsus was normal; in chewing, no discomfort or pain was felt. On the 22nd day the patient was discharged.

Case 2.—A female, 55 years of age, was admitted to the clinic complaining of a tumor in the nasopharynx and nasal cavity. The disease commenced two years ago with the blockage of the nose; about six months before, the nose was entirely obstructed; she began to breathe through the mouth; the right eye was watering; the right ear had been stuffed at times; there were shooting pains in both ears. The patient had been in the Novosibirsk Hospital, where a portion of the tumor had been removed, and she was then referred to the clinic of the University of Tomsk, where the following was found:

The general condition was satisfactory enough, the patient being a well nourished woman. Anterior rhinoscopy on the right: the nasal cavity occluded with the tumor; on the left, the tumor filled up the posterior part of the nasal cavity. The oral cavity: Carious teeth, many of them missing; tonsils not enlarged; tumor in the nasopharynx; the upper portion of the soft palate bulged forward; the tumor occupied the whole of the nasopharyngeal arch.

On the lower segment of the tumor, on the right, there was an ulcerated surface—the result of the previous operation. The tumor, immovable, bled when touched. Diagnosis: sarcoma.

On December 10, 1926, a Kocher operation under local anesthesia was made. It was found: the tumor from nasopharynx spread further; on the right it grew into the sinus maxillaris and completely filled it: the same condition was found on the left, the tumor filling up the upper portion of the sinus.

The patient bore the operation rather well; there were particular complaints of pain during the operative procedure. Ten days later she was given the common diet and began to eat solid food. Alveolar processes stood in the right manner; there was a slight mobility in them. On December 30 the patient was discharged.

Case 3.—K. J., a boy, aged 15, was admitted to the clinic November 12, 1926, complaining of the absence of nasal breathing as well as the swelling in the nasopharynx.

His father stated that two years before the boy had had a bad cold in the head, the nose became obstructed and at intervals bleeding from the nose occurred. Recently the bleeding has become more frequent and profuse; the nose was stuffed to such an extent that the patient ceased to breathe through it. The left eye commenced to water and bulged somewhat forward and outward. Drowsiness, apathy and excessive snoring appeared.

The patient was poorly developed for his age; anemic, with wandering glance; the left eye was watering, the mouth open. In the left buccal region, below the zygomatic arch, there was a bulging, disfiguring the face. The dorsum of the nose, in its bony portion, was enlarged towards the left side.

Anterior rhinoscopy on the left side: the nasal cavity filled up with the swelling extending as far as the cartilaginous part of the nose; the right choana was filled with the tumor, the lumen of the nose was free; the lower turbinal hypertrophied.

The oral cavity: several carious teeth, tonsils hypertrophied. At posterior rhinoscopy the following was observed: the nasopharyngeal cavity was filled with tumor, the choanæ covered with the tumor which filled the nasopharynx and originated in the nasopharyngeal arch.

Upon digital examination the movements communicated to the nasopharyngeal tumor were transmitted to the bulging in the region of the left cheek.

Diagnosis: Fibroma basis cranii accompanied with the ingrowth of the tumor into the fossa sphenopalatina and sinus maxillaris bilateralis.

On January 29, 1926, the Kocher operation under general anesthesia was performed, the posture of the head being according to Ross. When the nasopharynx was opened the following was observed: the tumor filled the whole of the arch; on the left side it grew as a branch process into the fossa sphenopalatina; on the right into the upper part of the sinus maxillaris.

The tumor was removed partly with a knife, partly with the strong forceps. In resecting the hard palate rather profuse bleeding occurred. Two weeks after the operation the patient received the common diet, was able to chew solid food and on the 20th day was discharged.

One year later the patient reappeared on account of the relapse of the tumor. The alveolar processes stood right; no mobility in them, the morsus was normal. The tumor of the nasopharynx attained the size of a small mandarin, had a knobby surface, was immovable, the choanæ were filled with the tumor.

Repetition of the Kocher operation under local anesthesia as before.

In resecting the alveolar processes and hard palate complete bony consolidation was found. The alveolar processes stood right; all teeth were preserved in full. In the resection of bones the bleeding was small. The tumor was completely removed.

Two weeks after the operation the patient ate solid food and on the 25th day was discharged as recovered. Alveolar processes were standing right; there was a slight mobility in them.

Case 4.—P. B., male, 18 years of age, was admitted to the clinic February 3, 1927, complaining of the absence of the nasal respiration and the tumor in the nasopharynx and nasal cavity.

The disease had appeared two years before, commencing with a severe cold in the head and bleeding from the nose. The nose had been gradually obstructed. One year before, the patient began to feel a swelling in the nasopharynx; his right eye was watering. The patient became pale, emaciated, lost his vigor and could not do any physical work.

The patient was poorly developed for his age, pale, nourishment poor. He complained of general debility, headaches and complete absence of breathing through the nose.

The anterior rhinoscopy: the right half of the nose was filled with a knobby tumor; the latter filled the whole of the lumen of the nose; the tumor grew into the sinus maxillaris; the left half of the nose in the region of the choana was filled with the tumor.

The oral cavity: a row of carious teeth, the tonsils small, with lacunæ. The soft palate displaced forwards and downwards; the nasopharynx filled with the tumor, as large as a big mandarin; the lower segment of the tumor stood out from under the soft palate; the tumor was knobby and of solid consistency, immovable and bled at the time of examination.

Diagnosis: Fibroma basis cranii.

On February 10, 1927, the Kocher operation, under local anesthesia. The tumor originated from the region of the choanal apertures; on the right the tumor grew into the sinus maxillaris; on the left it did so into the upper part of the sinus maxillaris.

The tumor was removed. Bleeding scant; the patient bore the operation well. A week following the operation, after the sutures joining the alveolar processes had been removed, the right half of the alveolar process began to drop down so that asymmetry ensued.

The patient was told to eat solid food and chew it up. Between the lower jaw and the dropping portion of the alveolar process the rubber plate had been inserted which the patient wore constantly, pressing it with his jaws. One week later the dropping half of the alveolar process stood at the level with the left half so that complete symmetry was regained. On the 23rd day the patient was discharged with a complete symmetry of the alveolar processes and the normal morsus of teeth (see photograph).

Thus the osteoplastic resection of the alveolar processes, according to Kocher, has been performed by me five times. In one case this was done under general chloroform anesthesia, and in four under local anesthesia.

The Technic of Local Anesthesia.—From the vestibule of the oral cavity, while raising the upper lip with the aid of retractors, the nervus infraorbitalis is anesthetised at the point of its exit from the foramen infraorbitalis. After this both the anterior and lateral surfaces of the upper jaw are anesthetised subperiosteally. Further, a long needle is inserted into the mucosa, immediately below the processus zygomaticus ossis maxillaris, subperiosteally the posterolateral surface of the maxilla is anesthetised.

After having obtained anesthesia here and without removing the needle the latter is directed into the fossa pterygopalatina and thence to the foramen rotundum; in this way the nervus maxillaris is anesthetized. On removing the needle a certain amount of the anesthetizing solution is injected into the cellular tissue of the fossa sphenopalatina.

After having anesthetized the maxilla superioris I pass to the anesthesia of the hard and the soft palate; here nervus palatinus is first anesthetized at the point of its exit from the foramen pterygopalatinum and finally nervus nasopalatinus is anesthetized at the point of its exit out of the canalis incisivus.

This somewhat complex anesthesia has been applied by me for two reasons: on the one hand, in order to obtain the most perfectly painless effect, and on the other, to bring about a decrease of bleeding at the time of the operation by means of anesthetizing at various points.

It would seem that the mere anesthesia of the main trunk of nervus maxillaris in the region of the foramen rotundum would suffice. This, however, is far from being so in reality; first, because one cannot attain the complete regional painlessness by this means, for such an extensive operation as that of Kocher's; secondly, because in anesthetizing merely nervus maxillaris in that region one cannot attain bloodlessness over the extensive operative field.

By means of the above anesthesia I was fortunate enough to have both these advantages at one time.

Anesthesia is performed either on one side first and then on the other, or alternately on both sides simultaneously; when using the latter method the anesthesia is more uniform. The amount of the anesthetizing solution (novocain 0.5 per cent with adrenalin) required for the complete anesthesia is 25 to 30 c.c.

The anesthesia in this case is complete; the patients bear the operation comparatively well, and it is possible to apply it to youngsters 15-18 years of age.

The technic of the operation is not so complex as it would seem at first glance. The trauma caused to the patient is of rather large extent, it is true, but necessary to provide a freer access to the tumor at its point of origin.

When the local anesthesia is done, after a pause of 10 to 15 minutes, the upper lip is raised upwards by means of retractors; it is not dissected along the median line as is the case in the typical Kocher operation, with one incision. The mucous membrane with the periosteum is dissected in the place of the transitory fold in the vestibule of the mouth from the level of a canine tooth on one side to the level of a canine tooth on the other.

Then, at the level of spina nasalis anterioris the broad chisel is fixed and alternately sometimes on one side, sometimes on another the alveolar processes are horizontally dissected, the mucosa of the oral cavity remaining uninjured. The boundary of the dissection of bones with a chisel should run beyond the eminence of the lower surface of the buccal process, but no farther, for in dissecting the bones farther the vessels and nerves which pass here can be injured.

After the dissection of the alveolar processes the latter, together with the hard palate, are displaced somewhat downwards and now with a slender sharp chisel the hard palate is dissected, together with the mucosa of the nose and hard palate from the median space between two middle incisors. Further, the soft palate is dissected up to its loose margin, the latter being left intact.

The nasal septum is displaced to one side or the other, and when necessary all of the nasal septum is resected as well as the lower turbinal. The separated alveolar processes, together with the palatal processes, are displaced outwards by means of sharp hooks, and ready access to the base of the tumor is secured, both sinuses maxillaris being exposed.

The tumor is removed under visual control, the nasopharynx is packed with a gauze plug. After the removal of the tumor, the separated alveolar processes are replaced into their previous positions and joined together with a silver wire, which is laid either about the two median incisors or all of them. The incisions of the mucosa and periosteum, as well as the soft palate, are sutured. Dressings support the inferior maxilla.

One of the chief reasons which impelled me to resort to this operation was the neglect of cases and the resulting extensive spreading of the tumors. The tumors in their growth did not confine themselves to the nasopharyngeal region and nasal cavity; in practically all the patients they grew into the neighboring regions. These tumors grew into the sinus maxillaris on both sides. In one case the tumor grew into the fossa sphenopalatina.

The gravity of these cases fully justified the choice of wide surgical intervention. The application of other methods in these cases, such as preliminary operations, operations of Langenbeck, Weber and Ollier, or the complete resection of the upper jaw, would not provide so ample an access to the tumor and especially to its extensions on both sides into the antrum maxillaris, ethmoid and fossa sphenopalatina.

Such a method of preliminary operation should be sought here which will at once expose not only the base of the tumor but also

the regions where its processes grew in; consequently, above all, the accessory nasal sinuses, including sinus sphenoidalis and fossa sphenopalatina.

Kocher's method best meets all these requirements; in separating alveolar processes the sinus maxillaris is widely open on both sides; in separating the hard palate the nasal cavity is widely open, where, upon the removal of the nasal septum, one is able to examine carefully the ethmoid, then the starting point of the tumor in the nasopharynx, the position of the tumor, its spreading over the fossa sphenopalatina—which certainly may not be obtained in any of the above mentioned methods since they are made on one side without giving free access to the other.

The cosmetic results are in favor of the Kocher operation. The scars on the hard and soft palate remain after this operation, it is true, but these are small; the patient is quite indifferent to them. As a result no outer signs are left after the operation, and this fact is of great importance for the patient.

The most valuable achievement of the Kocher operation is that it may be completely and successfully performed under local anesthesia.

When operating according to Kocher, apprehension may arise as to the viability of the resected alveolopalatal portions, and the integrity of teeth. This apprehension is the more natural when one considers the extensiveness of the trauma which is caused to the bones and mucous membrane of the upper jaw and hard palate. It is these apprehensions which render the Kocher operation unpopular.

In fact, these fears are much exaggerated as clinical experience has shown.

To make this clear I venture to map out briefly the scheme of the vascular system which supplies the region of the operative field.

The posterior part of the nasal cavity is supplied with blood through arteria sphenopalatina; sinus maxillaris is supplied through arteria alveolaris superior and arteria infraorbitalis; the hard palate and the tonsils are supplied through arteria palatina ascendens which is a branch of arteria carotis externa and through

arteria palatina descendens (a branch of arteria maxillaris interna).

The upper molar and canine teeth as well as gums are supplied through branches of arteria alveolaris superior; the incisors are supplied through branches of arteria infraorbitalis. The direction of the vessels is as follows: Arteria alveolar superior—posterior begins in fossa pterygopalatina, turns forward and downward, supplies the sinus maxillaris and runs to the upper molar teeth and their gums.

Arteria infraorbitalis emerges from the foramen infraorbitalis, branching as arteriæ alveolares superiores anteriores to the upper teeth and their gums. Arteria palatina descendens passes through the foramen pterygopalatinum and is divided into two arteriæ palatinæ minores, supplying the soft palate and neighboring parts, and arteriæ palatinæ majores supply the lower surface of the soft palate and gums.

Arteria palatina major in the region of canalis incisivus anastomoses with arteria nasalis posterior septi nasi; the latter artery is branched from arteria sphenopalatina.

Arteria palatina ascendens (usually a branch of arteria maxillaris externæ) sometimes begins from arteria carotis externa or arteria pharyngea ascendens; it supplies with blood the muscles of the soft palate, pharynx and mucous membrane.

Which of the above mentioned vessels are then inevitably injured at the operation?

In the resection of the hard palate these are, before all, the anastomosing arteriæ palatinæ majoris, with arteria nasalis posterioris septi nasi; and in the dissection of the alveolar process the upper dental arteries are injured—arteria alveolar superior anterior.

When operating according to Kocher, with such a minor impairment of large vessels, there is no ground to fear unhealing of the separated alveolopalatal processes; when well fitted to their usual places they heal well.

The continuous massage of the surfaces injured contributed best to the growing together of bones. The patients therefore are advised to take solid food as early as possible.

The postoperative treatment is not complex in these cases: it is reduced first to removal within 24 hours through the nasal cavity of the gauze plug from the nasopharynx, and then to the irrigations of the nasopharynx with a disinfecting solution through the nasal cavity.

CONCLUSIONS.

1. In the tumors of the nasopharyngeal tract growing into the adjoining regions: the accessory nasal sinuses, orbits and cranial cavity, especially where these ingrowths spread over both sides of the facial skeleton, the best operation is the Kocher method of osteoplastic resection of the alveolopalatinal processes of the upper maxilla.

2. The Kocher operation paves a broad way to the tumor, and enables us to reach its original point as well as to discern the arrangement of the processes growing into the neighboring cavities.

3. In operating according to Kocher the nasal accessory sinuses, especially sinus maxillaris, are widely opened, simultaneously on both sides, which enables us to set free both the processes of tumor grown into it as well as those grown into the orbit, sinus sphenoidalis and fossa sphenopalatina.

4. The Kocher operation in its technical procedure, in the amount of the blood lost, in its final results is much more advantageous to the patient than the temporary resection of the upper jaw, to say nothing of the complete resection.

5. The Kocher operation can be successfully performed under local anesthesia and is accompanied by a small loss of blood not only during the preliminary step, but when the tumor itself is removed.

6. The separated alveolopalatinal processes heal up rapidly, and the danger of nonhealing and the disturbance of the masticatory function of the upper jaw is to be feared least.

7. For healing the alveolar processes more rapidly the patients should as early as possible be given a common diet and invited to chew solid food and crack nuts.

8. The teeth do not suffer; it is true the patients feel, for some time, numbness, especially in the fore teeth, but this grad-

ually ceases. The teeth are not shed, and their masticatory function is not disturbed.

9. Cosmetic results of the Kocher operation are in its favor, no traces of the operation being left on the face, nor the facial relief being altered.*

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*Since the preparation of this article Prof. Nikolsky has encountered still another case of nasopharyngeal fibroma which has been successfully treated by the Kocher method.—Ed.

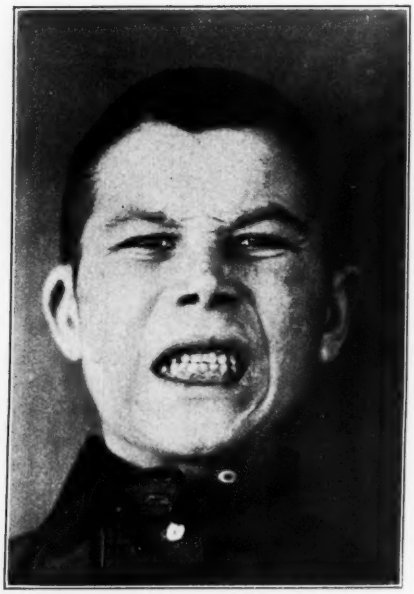

Fig. 1.—Case No. 4. Taken on the 23rd day after operation, showing the normal morsus of teeth of the upper jaw.

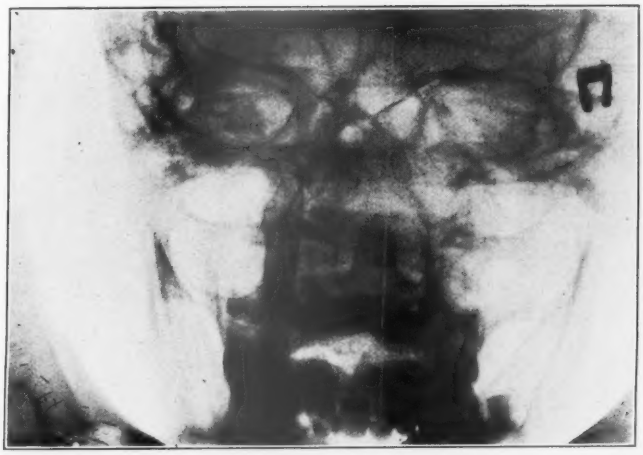

Fig. 2.—Roentgenogram of Case No. 5. The tumor filling the nasal cavity is seen, as well as the subcutaneous defect between the nostrils, the result of the previous operation.

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Fig. 3.—Roentgenogram of Case No. 5. Face. Taken after the contrasting mixture had been introduced into the upper portion of the nose; the mixture is particularly distinctly seen in the upper part of the nasal cavity above the tumor and in the lower portion of the tumor.

Fig. 4.—Roentgenogram of Case No. 5. Profile. Showing the point of the operative cutting off of the alveolar processes; the silver wire is seen uniting both halves of the alveolar processes.

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Fig. 5.—Roentgenogram. Taken after the removal of the tumor: the lumen of the nasal cavity is seen.

Fig. 6.—Photo of Case No. 5. Taken on the 20th day after operation.

LV.

NASAL ALLERGY AS RELATED TO HAY FEVER AND HYPERESTHETIC RHINITIS.*

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The symptom-complex of periodic attacks of sneezing, watery discharge from the nose, itching and nasal obstruction is a familiar one to the rhinologist and needs no description. Because of its disputed etiology and pathology this syndrome is designated by various names, such as hyperesthetic rhinitis, vasomotor rhinitis, perennial rhinitis, allergic rhinitis, atopic coryza and hay fever.

The importance of this syndrome is based on a number of factors. In the first place, the symptoms may be very annoying and disturbing to the equilibrium of the nervous system, and may even interfere with the daily tasks of the individual. The significance of this is perhaps better appreciated when we consider that from 1 to 2 per cent of the American population are handicapped by this disease, and some of them more or less throughout the year. Perhaps more important yet is the tendency to certain complications. In about 6 per cent of seasonal hay fever cases and in 25 per cent or more of all year round hay fever cases there are found sinus disease which are usually a direct result of the edema of the mucous membranes or lack of ventilation of the sinuses. About 30 to 40 per cent of pollen hay fever cases are complicated by seasonal asthma, many of which later terminate into chronic year round asthma. To put it very graphically, we are confronted here in Chicago with a problem of about 50,000 cases of hay fever and hyperesthetic rhinitis, 15,000 of

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which already have seasonal asthma, and about two or three thousand chronic asthma as a direct result. In addition, due to the same causes, we have about three or four thousand cases of chronic sinus infection to contend with.

Whether we maintain that this is purely a problem for the rhinologist or whether we argue that this is a field for the internist, there is no argument that this enormous responsibility must be shouldered by someone.

ETIOLOGY.

Although it must be admitted that the concept on which many of us base the management of the hypersensitive nose is far from perfect and leaves much room for failure, we contend, nevertheless, that the following of this concept will yield one considerable success. On the basis of our observations and accumulated data of workers in this field in the last fifteen years there is very little doubt that the concept of allergy in relation to this group of conditions has come to stay.

By the term allergy I mean the pathologic hypersensitiveness of body tissues to a substance which is harmless to the majority of individuals. This substance is usually in the form of a protein but it probably occurs in other forms.

In this syndrome of hypersensitive rhinitis in a large proportion of the cases we are capable of demonstrating that the contact of certain substances with the nasal mucosa act as irritants producing the pathologic changes and the well known symptoms. It is not denied that in some cases other factors may be demonstrated, such as disturbed endocrine balance, particularly myxedema, and that in many cases no allergy can be demonstrated. However, there is no single concept which explains the pathogenesis of this disease as consistently as allergy.

In the production of allergic manifestations, three factors may be involved. First, there must be a certain constitutional difference in the allergic individual which allows him to become sensitized. Although most of us are exposed to feather dust, for example, only a small proportion of us become sensitive to it. This

constitutional abnormality is hereditary in at least more than half of the cases. The second essential in the production of symptoms is the presence of the specific exciting substance, such as feather dust. Without that, although sensitization is present, no clinical manifestations will result. In some cases at all times, and in some at various intervals, a third factor may be necessary before the individual will have clinical manifestations of hyperesthetic rhinitis. This may be termed the nonspecific exciting cause, and consists of such factors as mechanical (septum, polyps, dust); thermal (cold air); neurogenic (nerve strain, etc.); chemical; and infectious (sinusitis, etc.). These latter phenomena may be sufficient to disturb the balance of what is known as "allergic equilibrium." It must be emphasized that the admission of existence of such nonspecific elements in this scheme does not detract from the allergic viewpoint but rather broadens our conception and aids us in managing our patients more rationally and more successfully.

The specific exciting cause of this allergy may fall into one of several groups: pollens, animal epiderms, foods, bacteria and certain miscellaneous substances. The pollens constitute the most frequent cause for these nasal symptoms. It should be remembered in this connection that it is only those pollens that are carried by the wind that can cause hay fever. Plants of distinct odor or bright color, such as goldenrod, roses, and white clover, are pollinated by insects and therefore are not ordinarily a source of hay fever.

In the Chicago district there are three principal hay fever seasons. Any individual may have symptoms in one or all of these periods. The first season occurs chiefly in March, April and May, and is due to the pollens of the trees. This form of hay fever is usually very mild, of short periods but frequently recurring, and usually diagnosed both by the patient and physician as "spring colds." The second season begins the latter part of May and continues through June and July; this is caused by pollens of the common grasses, principally orchard grass, June grass, timothy and redtop grass. The third and most important season begins about the middle of August and continues until the

cold weather. This is caused principally by the giant and short ragweeds and burweed marsh elder. In addition to the above, there are other plants which can be definitely incriminated at times.

The all year round hyperesthetic rhinitis of the allergic type is caused usually by epidermal proteins, foods, bacteria and miscellaneous substances. The epidermal substances constitute the most common cause of hyperesthetic rhinitis. In this group the feathers are the most important single item. Many cases of hyperesthetic rhinitis owe their trouble to their feather pillow or down comforter or down in the furniture. This contact may also occur directly from the fowl, such as in farmers and butchers. Occasionally allergic rhinitis or asthma will result from contact with the feathers of the less common type. For example, I have under observation an individual who is sensitive to canary feathers.

Cat hair and dog hair have been responsible for many instances of hyperesthetic rhinitis. In this connection it is interesting to note that the first description of cases of asthma and hyperesthetic rhinitis due to animal emanations was given by Hyde Salter in 1859 in his memorable book. Salter himself was a sufferer from cat asthma and his remarkably clear clinical observations are worthy of reading. It is not always essential to have a cat or dog in the home in order to be getting hyperesthetic rhinitis from their hairs. Frequently a neighbor's cat or dog, or cat fur disguised under a more expensive name, may be the source of the trouble. Other furs may be a source of hyperesthetic rhinitis. The rather common custom of naming furs by names other than the animals from which they originate is a source of confusion not only to the woman who purchases a fur coat or collar but to the allergist as well. For example, the sable or fox fur is probably dyed hare; the ermine that milady is wearing may be only white rabbit skins; and the bear or leopard skin most likely came from the lowly goat.

Horse hair or its dander is quite a common cause of sensitization in hyperesthetic rhinitis, even in cosmopolitan Chicago. Horseback riding, horse hair in the mattress, furniture stuffed

with horse hair are frequent sources of this type of sensitiveness. However, horse dander is so extremely toxic to some cases of hyperesthetic rhinitis that the minute quantities present on the street in the air may be sufficient to be almost a constant source of annoyance. Cattle hair is at times a cause of irritation. This hair is commonly found in mattresses, furniture and in felt padding. In a recent case of cattle hair sensitiveness the source of the hair was located in the felt padding under the carpeting. With the removal of this padding immediate improvement in the patient's symptoms resulted. Other epidermals that may be causative factors are rabbit hair, hog hair, and others of lesser importance.

Foods are also a fairly frequent source of perennial rhinitis. This sensitization is not confined to any food or any group of foods—almost any food may cause it. Among some of the foods that I have encountered in my practice as causes of nasal irritation are wheat, bananas, potatoes, chocolate, peppers, mustard, celery, lettuce, beans, peas, milk and eggs. Not uncommonly food sensitization in these cases is a complication secondary to other sensitization such as epidermal proteins or pollens.

It is still an unsettled question whether allergic reaction may result from bacterial proteins. There are cases in which no positive allergic tests can be demonstrated and in which considerable bacterial infection seems to be present. Bacterial infection is often also a complicating feature in nasal sensitization, tending to maintain the irritability of the nasal mucosa after the primary allergic cause has been removed.

There are a number of miscellaneous substances causing hyperesthetic rhinitis. Orris root powder, which is a common ingredient of many face powders, talcum powders and other cosmetics, is perhaps the most important of these. In some cases of orris root sensitization the tolerance is so low that the inhalation of the small amounts of powder from the skins of others or the powder in the air, such as in theaters, washrooms, etc., may be sufficient to cause considerable nasal irritation.

Cottonseed is also a fairly frequent cause of this trouble. This material is usually to be found in the dried state in mattress cotton

or in upholstered furniture. Cottonseed oil used in cooking and soaps may also be a source of trouble. Pyrethrum, used in insecticides; kapok, lycopodium and other substances have also been incriminated. House dust, aside from its mechanical action, often has a specific allergic action on the nasal mucosa. In addition to the above, certain drugs have been observed to cause hyperesthetic rhinitis in some individuals; the most common of these are aspirin, phenacetin, pyramidon, quinine and ipecac.

Although it is ordinarily held that the seasonal cases are caused by pollen and the nonseasonal cases by the other irritants, this is by no means always true. Seasonal hay fever is not always due to pollen, as I have shown elsewhere. Rhinitis may occur only in summer because of horseback riding during that season or because of eating certain seasonal foods. Two instances of seasonal "hay fever" that I encountered last summer,[1] in whom the attacks have occurred for seven or eight seasons, were due to orris root in cosmetics and not to pollen. In such cases it is assumed that the excessive use of powders in the summer exceeded the tolerance point of those individuals.

On the other hand, irritants exclusive of pollen to which individuals are sensitive may not be sufficient to produce symptoms at any other time except the pollen season. In such instances, in addition to the hay fever treatment it may be necessary to avoid certain foods or other substances during the pollen season in order to obtain satisfactory results.

DIAGNOSIS.

The diagnosis of hyperesthetic rhinitis is easily made on the characteristic symptomatology and the appearance of the nasal mucous membrane. It is extremely important, however, to endeavor to make a specific diagnosis—to determine the actual exciting substance in the particular case. This is done usually by skin tests. Linear abrasions are made, most commonly on the forearm or upper back, and extracts of the materials in question are dissolved with N/10 sodium hydroxide. In some cases liquid extracts are used. Positive tests are usually evident within five to twenty minutes and consist of an urticarial formation with

surrounding erythema and usually accompanied by itching. In some instances these reactions may be delayed in appearance to a few hours or even twenty-four hours. In some cases the cutaneous tests are negative and we must resort to other forms of testing, such as intracutaneous, conjunctival or nasal. This signifies that although the conjunctival or nasal mucosa is sensitive to the substance the skin is not necessarily so.

At this point it should be emphasized that the practice of making a few tests is entirely insufficient, misleading and worse than no tests at all. The individual should be tested for all substances with which he may possibly come in contact by inhalation or ingestion—and this may mean 150 tests or more. Those omitted may contain the very substance for which one is looking. Group tests, in which several materials are combined in one group for the purpose of reducing the number of tests, are only mentioned to be condemned: they are unscientific and unreliable.

There are many variable factors in the production of skin reactions, such as the variability of the individual's skin, the difference in degree of reactivity of various proteins, the possibility of negative or refractive phases, and borderline reactions. It can thus be readily understood that the interpretation of these tests require considerable experience and judgment.

Positive reactions are obtained to the pollens in about 99 per cent of the seasonal hay fever cases, and reactions to other proteins in approximately 30 to 50 per cent of the cases of hyperesthetic rhinitis. In this connection it should be mentioned that the presence of sensitization to a particular substance does not always result in a positive skin test; this is especially true of foods. It is likewise true that the mere presence of a positive skin test does not indicate necessarily that the individual is having clinical symptoms at the time from that particular substance.

Under all circumstances clinical and therapeutic trials must be instituted to make or to confirm the diagnosis. This means, for example, that in the case of a positive test to cat hair the patient should be made to fondle a cat, or in the case of horse dander reaction to go into a stable for a few minutes. The resulting almost immediate onset of symptoms is quite convincing.

In the cases of food sensitiveness either elimination of the suspected food or the addition of it to the diet should further corroborate the diagnosis.

Cases of seasonal hay fever which have not previously responded well to pollen treatment have often been found sensitive to other proteins, such as foods and inhalants, in addition. Attention to these extra-pollen factors seem to further cut down the number of these failures. These patients, although sensitive to these substances, apparently may have very little or no symptoms from them the major part of the time. However, the irritation of the nose by the pollen may enhance the action of one upon the other. In the last year or two it has been my custom, therefore, to test, whenever possible, all hay fever cases with foods, epidermals and miscellaneous substances, in addition to the pollens.[2]

TREATMENT.

The treatment of the hay fever or hyperesthetic rhinitis patient may be discussed under several headings. Specific treatment aims at elimination of the offending agent, or densitization to it if the former is not feasible. Those of us who do allergy work are constantly being criticised on the basis that such treatment is not fundamental; that it leaves the allergic individual still constitutionally hypersensitive, and easily capable of acquiring other sensitizations. I admit that all this is true; that it would be much more basic to alter the constitutional characteristics of the individual so that he will never become sensitive to anything. However, in the present state of our knowledge this is not possible and we do the next best thing—treatment aimed at the exciting cause.

Whenever possible, sources of sensitization should be eliminated. This is usually feasible in the case of almost all foods, feathers, cats, dogs, etc. For this object it is important to have knowledge of the location of these substances and also to remove them thoroughly.[3] For example, it may be necessary to remove feathers from the entire household or to remove the horse hair mattress in addition to warning the patient against horseback riding. It may be necessary to get rid of an oriental rug

containing goat hair or carpet padding containing cattle hair. It is important to realize that when the patient is sensitive to wheat not only is he to avoid white bread, cake, cookies, pie, spaghetti, etc., but also gravies and "rye" bread.

In cases of sensitization to pollens, horse dander, cottonseed, orris root and house dust and occasionally other substances, elimination is usually unsuccessful and desensitization must be instituted. The latter consists usually of subcutaneous injections of the offending protein in increasing amounts. The intervals between treatments, the initial doses, and the rate of increase in doses are best gauged by the patient's individual reaction. At times other methods of desensitization are employed, such as intradermal injections, nasal applications or oral feeding. It is impossible in a paper of this scope to give detailed directions for treatment.

The average hay fever patient should begin treatment about ten or twelve weeks prior to his pollen season. In many cases this time interval may be modified considerably. A patient seen about two weeks prior to his pollen season will be given injections once or twice daily perhaps. In instances in which the individual is unable to come to the office frequently treatments are begun much earlier than the customary time and given at longer intervals. In many cases I have found it advisable to employ all year round treatment for hay fever, giving an injection once in every two or three weeks. This is especially applicable to those cases who have had the usual course of preseasonal treatment. The preseasonal treatment is extended also into part of the pollen season. Bacterial vaccines are often used also as a supplementary measure during the season.

The results of hay fever treatment have been good on the whole. In about 85 to 90 per cent of the cases there has resulted satisfactory relief for the season. In most cases the treatment must be repeated the following seasons; and with each successive year of treatment better results are obtained. Many patients reach a point where they no longer need treatment.

The factors enabling one to change a low percentage of good results to a situation whereby 85 per cent or more of his patients

derive relief from their hay fever, may be summarized as follows:[4]

1. Early preseasonal, combined with seasonal pollen treatment.

2. Individualization of dosages of pollen. The drug store package of standardized doses fits very few individuals. Some patients are a hundred times as sensitive as others. To treat both with the same doses is irrational practice.

3. An attempt to reach a greater maximum dose in the majority of the cases than is usually the custom.

4. The careful determination of the species of pollen to be used.

5. The preservation of the potency of the pollen by refrigeration in concentrated form.

6. The testing of the patient with substances other than pollen, as the former may be a marked contributing factor, or even the sole cause, of the hay fever symptoms.

In the cases of perennial hyperesthetic rhinitis the results of treatment are usually successful in those instances in which definite allergic factors have been determined. It is important to realize, however, that most of these cases are complicated by multiple sensitizations. It is, therefore, almost impossible to make a rigid outline of the individual's management of treatment immediately after the tests are completed. It is frequently necessary to modify or even change entirely the mode of treatment during the course of observation of the patient. It must be frankly admitted that the procedure of diagnosis by the allergist and subsequent allergic management by one who is not versed or interested in the intricacies of allergy, is not productive of a high degree of success. This sort of predicament may be likened to what we might expect if the rhinologist, after making a diagnosis of sinus infection, would refer the patient to the internist or allergist for treatment.

Palliative treatment has to be used frequently. Ephedrin orally or by nasal instillation or spray is the drug of choice in hay fever. In hyperesthetic rhinitis belladonna gives considerable relief in many cases. I have been thoroughly disappointed in the use of calcium in these conditions, nor have I found any distinct deviation of the blood calcium from the normal. The use of

thyroid plus calcium, or parathyroid plus calcium, has been given a thorough trial in our allergic clinic on a large series of cases several years ago—with almost totally negative results. Neither have I found any definite relation of these allergic diseases to thyroid disturbances as evidenced by the basal metabolic rate. The recently advocated use of nitrohydrochloric acid internally for hay fever has been, in my hands, almost a total failure in the last two seasons in a fairly large series of cases. I have had no experience with the use of the ultraviolet light applied intranasally, but general body radiation I know to be of little benefit in hay fever or hyperesthetic rhinitis.

In cases of hyperesthetic rhinitis in whom no evidence of sensitization is present, vaccine treatment seems to give the greatest hope for relief. However, the percentage of good results in this group is not great by any method of treatment. In an occasional case I have obtained good results with tuberculin.

The majority of cases are not helped by change of climate. When relief does occur it is usually due to change of diet or home surroundings. However, in pure pollen cases a residence in a climate free from the particular plants to which the individual is sensitive will give relief.

The field of local nasal treatment belongs to the rhinologist and I care to say very little about it. My impression from rhinologists of my acquaintance indicates that they have found such local treatment not very satisfactory as a whole.

The question of operative treatment of the nose is a much discussed one and I hope will be discussed more at length this evening. I can say without fear of contradiction that there is too much nasal surgery done in allergic conditions and that in many cases the allergic nose or asthma is aggravated by the surgery. I think rhinologists of experience now agree that there should be clear cut indications for surgical interference in the allergic nose. Definite sinus infection must be taken care of; polypi should be removed; other nasal pathology must be treated surgically at times. However, there are too many turbinectomies, septum operations and ethmoid exenterations. On the other hand, I must also confess that many men in the field of allergy are apt

to disregard local conditions in the nose almost entirely. I am sure that this is also an unjustifiable attitude. There should be closer cooperation between the allergist and rhinologist. Not only will the individual patient profit by such cooperation but advancement in knowledge of this common disorder will be more rapid. In my own practice I make it a rule to have every case of asthma or hyperesthetic rhinitis examined by a competent rhinologist.

The cases of nonpollen hyperesthetic rhinitis at the present time must be divided into two groups, the allergic and the nonallergic. The importance of classifying any one individual is apparent from what has already been said about the treatment of the allergic cases. At the present time we must rely almost exclusively on sensitization tests to determine this point. From the literature and from impressions gained in discussion with rhinologists it seems that the latter are unwilling to give an opinion whether they are dealing with the allergic or nonallergic type by the appearance of the nasal structures. A point that I have found useful, and is also discussed by Balyeat,[5] is the observation that in allergic rhinitis there is usually present the sensation of itching either in the nose, pharynx, palate, ears or eyes. In the nonallergic cases itching is usually absent.

The high percentage of cases of hyperesthetic rhinitis in which allergy cannot be demonstrated increases our therapeutic failures considerably. Whether we are dealing in such instances with an unknown allergic cause or with an entirely different pathologic entity is an unsettled question. It is possible that just as cough and dyspnea are not necessarily symptoms of allergic asthma, or even bronchial asthma of any origin—so sneezing and rhinitis may be merely symptoms representing more than one clinical entity. What we ordinarily now term hyperesthetic rhinitis may presumably be classified into two or more clinical conditions, the cause of which may be entirely dissimilar. Thus it becomes further evident that for the sake of progress in this field there should be close cooperation between the allergist and rhinologist and a combined effort on the part of both to solve this important problem.

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LVI.

RATIONAL OPERATIVE PROCEDURES FOR CHRONIC HYPERPLASTIC SINUSITIS IN ASTHMA: A PRELIMINARY REPORT.*

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There are numerous articles in current medical print dealing with allergy and asthma, but there still remains an appreciable group of asthmatics whose problems have not been solved. We shall discuss some personal experiences in the application of radical operative procedures to a selected group of incapacitated asthma patients with chronic hyperplastic paranasal sinusitis whose asthma had persisted and increased in severity under accepted methods of treatment.

Asthma is a symptom complex of widely varying intensity and causation. In many cases the symptoms are evanescent or mild and succumb to a sensibly regulated hygienic and dietetic regime with possibly an occasional injection of epinephrin. When on the basis of sensitization tests, spectacular cures follow the throwing of a cat, dog, furs or feather pillows out of the house, the physician may be pardoned a nonchalant Sherlock Holmes sense of pride. There is, however, a small group of asthmatics whose struggles for breath are almost constant and whose sufferings are terrible. Only by increasing doses of epinephrin hypodermically are they able to breathe. Their condition is pitiful in the extreme and death not uncommonly eventuates. Sensitization tests do not give decisive information, and when positive reactions occur the elimination of food or contact allergens fails to influence the

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asthma except for a brief period if at all. Careful histories elicit no clues save possibly a history of repeated upper respiratory infections with exacerbations of asthma with each new infection. Sometimes a close relationship of "flu" and "colds" to the beginnings of the asthma may be developed. Often they have had repeated intranasal operations. It is in such cases that an exhaustive rhinologic study almost always reveals a chronic hyperplastic sinusitis or pansinusitis.

For clinical purposes we have divided our asthmatics into two groups, (1) allergic or exogenous, i. e., asthma from pollens, food (all ingested allergens), emanations (animal, dust, chemical, etc.); (2) bacterial or endogenous. Although bacterial asthmas are commonly assumed to be allergic in character, yet they differ from other asthmas in the fact that the asthma producing excitants are living and reside in the tissues of the patient. It is logical then, to assume that a cure of such asthmas will best be accomplished by as complete excision as possible of responsible bacterial infected tissues.

With this assumption as a working hypothesis in selected cases of severe bacterial asthma we have applied the technics developed for removal of hyperplastic sinusitic mucosa en masse.

Patients coming to us with severe asthma are subjected to a detailed medical survey, often with hospitalization for observation, study and preparation for operative attack. Systematic sensitization tests are carried out when indicated. Elimination diets are commonly used. Painstaking efforts are made to elicit any possible environmental allergen sources. We have commonly found such investigations to be of greater value than sensitization tests. If the source of the asthma lies in the sinuses the general clinical survey will invariably place it there and the rhinologic examination confirm it.

Rhinologic studies must be systematic and detailed. The failure to locate an ethmoid or sphenoid with a hyperplastic membrane may mean failure. The presence or absence of purulent or mucoid secretion in the nose is immaterial. Nasal polypi or polypoid degeneration of the turbinate bodies may be present but their absence is not conclusive. It is the chronically infected tissue lining and more or less completely filling the sinuses with its polypoid

hyperplasia that we believe has been responsible for the asthma in the cases which we are reporting. Obviously, radiography is the most important local diagnostic measure. The radiologist must be especially trained in sinus work. His equipment must be of the best, his technic so standardized that were the patient to return six months later radiograms taken in the same position have approximately the same photographic densities. Above all he must be a good photographer. We have adopted the technic and positions of Proetz[1] and Baum.[2]

If the ethmoids or sphenoids are suspected and the nasal examination does not show such gross pathology as to render it unnecessary, lipiodol is used to outline them by Proetz's method. The antra are filled with lipiodol by introducing a catheter through the natural ostium. After having seen the interior of these sinuses, I consider the puncturing of the antral wall with a needle or other sharp instrument unreliable and dangerous, as they are frequently completely filled with hyperplastic and polypoid degenerated tissue. The frontal sinuses may be filled by Proetz's method if the diagnosis is in doubt, but in most cases the radiograms show them either clear or so dense as to leave no doubt as to their condition. The infected antrum may be secondary to dental infection, and in that case there is frequently no pathology seen in the nose other than a chronic hypertrophy of the turbinate bones of the affected side.

Up to the present time all operations have been done under local anesthesia, preceded by morphin and scopolamin. There have been no deaths in this series, although some of those operated upon have been very poor operative risks. Hypodermics of epinephrin and caffeine sodium benzoate are prepared for emergencies and have been used. The only postoperative complication has been erysipelas in an elderly lady on the seventh day following an antrum operation.

The operation consists in as clean an excision as possible of all diseased tissue. This means in chronic hyperplastic pansinusitis an external operation of the antrum, frontal sinuses, ethmoids and sphenoids, with removal of as much of the mucous membrane intact as possible; removal of the middle turbinates if they have undergone polypoid degeneration must be done. If the infection

involves only one sinus, as an antrum from a tooth, only that antrum requires attention. However, it must be an external operation, such as the Caldwell-Luc, and the diseased tissue in the alveolar process completely removed at the same time. The ethmoids offer especial difficulties for complete removal of the infected tissue. The sphenoid in certain cases has extensive and irregular cells which are most difficult to reach. The importance of the sphenoid is well illustrated by two cases in which I did not excise the mucous membrane. Both were much improved but continued to have some asthma. One of these returned and the mucous membrane of the sphenoids was removed with prompt cessation of the asthma. An unsuccessful attempt was made to relieve this patient by a vaccine made from secretion from the sphenoids—a very minute dose caused a most violent asthmatic reaction. I have used Lynch's operation on the frontal, ethmoid and sphenoid, and have found it very satisfactory. The postoperative results on the nose have been amazingly good. If the operation has been complete there is no scabbing and seldom any return of polypi. The patients have not complained of dryness.

While the operative procedure sounds formidable, we beg to call your attention to the following considerations: We have limited our experiments to adults who have not been relieved by other means and who were incapacitated. All of them had tried to obtain relief in many ways, and many of them had been subjected to one or more nasal operations. No operation has been done except for the removal of chronically infected membrane. This work was only undertaken after we had suffered many disappointments from incomplete excision of "bacterial membranes." We have reoperated some of our previous failures and had success as a reward for our perseverance.

The results of en masse excisions of hyperplastic infected membranes on severe asthmas are often brilliant. Patients who have been sitting up struggling for breath, asking repeatedly for hypodermics of epinephrin, within 24 to 48 hours require no epinephrin and sleep comfortably on one pillow. The expiratory dyspnea and the fixation of the chest in forced inspiration lessen rapidly and respiratory elasticity is restored. The dry rales and squeaks disappear rapidly and the lung findings after several

weeks have disappeared entirely or are minimal. Dr. A. B. Smith, whose paper appears in THE ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, December, 1929, says: "The author feels certain that complete removal of all of the infection in these areas in from 70 to 80 per cent of the cases would completely cure the patient." Our results coincide with his conclusion.

An illustrative case was that of an elderly woman who had suffered from asthma for twenty years. Her asthma had recently increased in severity and had become life threatening. A careful history elicited the interesting fact that the onset of the asthma had just preceded extensive dental work involving many of her upper teeth. The patient volunteered the information that her teeth were very bad at that time. Changes of climate, hygienic, dietetic and medical regime had been instituted, but during the last two years the asthma had become practically constant and was controlled only by several hypodermics of epinephrin daily. Signs of impending heart failure were present. There were no abnormal secretions in the nose and only a chronic tumefaction of the left inferior turbinate was found. Radiograms showed only a moderate increase in density at the base of the left maxillary, which lipiodol confirmed as polypoid. Teeth films revealed marked alveolar disease in the left side with quite evident involvement of the antral floor of dental origin. A combined alveolectomy and Caldwell-Luc operation was performed with complete removal of all the alveolar disease, the floor of the maxillary and lining membrane. Only one hypodermic of epinephrin was used after the operation. The patient was asthma free within 24 hours and has remained so to date (three months). The history and the radiograms determined the sinusitis and asthma in this case to be dental in origin. The operation and the subsequent clinical course have confirmed it.

An examination of the bacterial membranes taken from all cases showed a very marked departure from the normal. The mucosa was one-eighth to one-fourth of an inch in thickness and in addition there were usually polypi. In some cases only a few polypi were present while in others the whole surface of the membrane was covered by them like a carpet. It is difficult to conceive how treatment could restore such membranes to

normal, and it is equally evident that irrigation through the natural ostium is useless. Intranasal drainage through a window under the inferior turbinate will be mechanically obstructed by these thick membranes and polypi. Microscopic examinations of the specimens show such extensive degeneration as to render the probability of a return to healthy membrane very problematical. Note in the illustration the loss of ciliated epithelium and the cystic degeneration. In addition, although not shown here, the whole membrane is invaded by bacteria. Kirstner[3] showed this to the Section of Rhinology of the American Medical Association in Portland last year. To attempt to treat a chronically infected mucous membrane of this type by making a larger hole in the sinus and irrigation is analogous to treating a chronically infected tonsil by local application.

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No. 1. Radiograph showing rounded area of increased density in the floor of both antra.

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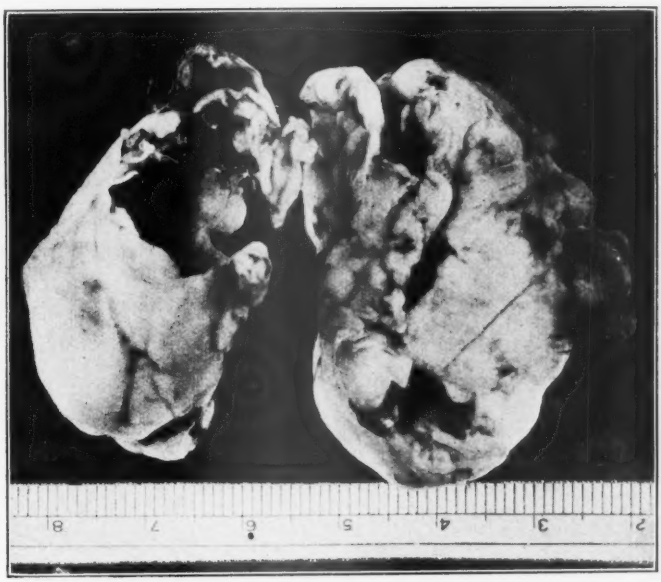
No. 2. Lipiodol in antra showing marked filling defect of right antrum and less defect in left antrum corresponding to shadows in radiograph.



No. 3. Gross specimen showing markedly thickened membrane and polypi.

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No. 4. Greatly thickened mucosa with marked cystic degeneration. Ciliated mucous membrane largely destroyed with only occasional patches remaining.

LVII.

SOME PRACTICAL POINTS IN THE USE OF ELECTRO-SURGICAL MEASURES IN THE UPPER RESPIRATORY TRACT.*

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PHILADELPHIA.

During the past twenty years, in which high frequency electrical currents have been used in the treatment of surgical conditions, their field of usefulness has been gradually extended. The simplicity of their application and the results obtained from their use in surgery in general have placed them in a position of considerable importance.

In this communication we shall not concern ourselves with the mode of their production but only with their effects upon certain nose and throat conditions.

D'Arsonval, professor of physics in the College of France, Paris, was the first to show, in 1890, that when alternating electric currents used to stimulate muscle contraction were increased in frequency to 5,000 and more per second, these contractions gradually became feebler and finally ceased altogether. In February, 1891, he reported that he was able to pass high frequency currents through his own body with no reaction and no sensation other than warmth.[1] The frequencies of the current used at present for surgical purposes are from 500,000 to 3,000,000 per second.

Nagleschmidt[2] and Doyen[3] were the first to apply these currents to any great extent to medical and surgical conditions and reported their early results in 1907 and 1909, respectively.

Lewis Jones was the first to introduce the use of high frequency currents for the treatment of medical conditions into

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†From the Department of Otolaryngology of the University of Pennsylvania.

England in 1909. In 1910, Nagleschmidt, at the invitation of Jones, went to St. Bartholomew's Hospital in London, where he successfully removed a large pedunculated tumor from the nasopharynx by this means. Since this time numerous reports of the use of this method in the treatment of nose and throat conditions have come from authors in Europe and America.

Recently, Cushing[4] of Boston applied the term electrosurgery to the use of the three modalities which are used at present, namely, electrodesiccating currents, electrocoagulating currents and cutting currents.

The electrodesiccating current is known as the Oudin current and formerly was called the fulgurating current. Its action is dehydration of the surface of tissues by showers of sparks from a single, usually sharp pointed, electrode. This electrode is not brought in contact with the tissue to be destroyed but is held a short distance from it, thus permitting the sparks to jump across the intervening air space.

The intensity of these sparks may be increased or diminished, so that a corresponding increased or decreased amount of dehydration may result. This method is not well adapted to deep and narrow orifices and can be used to best advantage in surface lesions or in wide open cavities. More scar tissue follows its use than that of the D'Arsonval current.

The electrocoagulation or D'Arsonval current and the more powerful cutting current are both dependent upon the use of two electrodes—a large or indifferent electrode, usually a thin sheet of lead about 8 by 12 inches in size, and a small active electrode which may vary in size from a large darning needle to a small sewing needle.

In the treatment of nose and throat conditions, the indifferent electrode is placed over the mid-dorsal region, thus causing the current to pass through a fairly large mass of body tissue in order to reach the point of application of the small electrode. The body tissues between these two points offer a certain amount of resistance to the flow of these currents, with the result that heat is produced. This heat is greatest in the region of the maximum density of current, which is in the vicinity of the small

electrode. The small amount of heat produced in the region of the large electrode is quickly dissipated by the blood stream, but the large amount of heat about the region of the small electrode, which usually is inserted into the tissue about one-fourth of an inch, causes the tissue to coagulate to a greater or lesser depth, depending upon the strength of the current and the length of time of application.

If a comparatively strong current is used for a short space of time, the area of coagulation remains close to the active electrode. If deep coagulation is required, a weaker current is necessary. This permits the longer time of exposure to bring about the desired result before charring occurs about the electrode with a consequent cessation of current.

Cutting is accomplished with a current whose to and fro movements are as many as three millions per second. The active electrode is of necessity as thin as possible, yet it must be rigid if it is to be used as a knife. A thin steel sewing needle may be used for this purpose, and it can be moved readily through the tissue, dividing it almost as rapidly as would a sharp scalpel.

The cut surface of this divided tissue has the appearance of being seared, although the electrode itself does not become hot. Wounds of this sort may be sutured and healing takes place by first intention, since the depth of coagulation is only a fraction of a millimeter. Deeper coagulation may be obtained with this current when its intensity is reduced.

TECHNIC AND ELECTRODES.

The proper machine setting is very important before beginning any operation, and a foot switch is absolutely necessary so that the current is under control of the operator at all times.

The choice of anesthesia depends somewhat on the size and location of the lesion to be treated. In general, small growths and superficial lesions of the mucous membrane of the nose and throat may be anesthetized with 10 per cent cocain solution or 1 per cent novocain solution. Larger growths involving deeper structures demand a general anesthesia. Gas or chloroform should be used. Ether, being inflammable, should be avoided. However,

Tobey[5] of Boston states: "The danger of ether explosion seems to be entirely obviated by the complete removal of all ether from the vicinity of the patient and waiting of at least three minutes before proceeding with the application of the current."

A pencil shaped handle made of hard rubber is the most handy for holding the electrodes most frequently used in the nose and throat.

The tips of the electrodes vary in shape, depending on the kind of work to be done. They should be insulated throughout their length, with only the tip exposed. The tips may be either pointed steel bars, straight or curved, or thin steel wire loops. The loops are useful in removing bits of tissue for diagnosis.

The Krause nasal snare can be made into a very good electrode and handle by slipping a thin rubber tube over the canula of the snare. In using this instrument it is essential that the operator wear rubber gloves.

The advantage of the combination of snare and cutting or coagulating current is very great in the removal of vascular growths of the nasal cavity.

EFFECTS OF ELECTROCOAGULATION.

The desired effect of coagulation is obtained when a snow white circle appears on the tissue about the electrode. For tonsil destruction (for example), the machine should be so set that this circle should have a diameter of about one-fourth of an inch after about one second's application of the current. When such current is applied longer than the time required to simply produce this snow white area of coagulation, charring occurs about the electrode with a sparking effect. If the application of the current is further continued, crackling occurs, as of a sudden escape of steam, and the tissue balloons up, giving the impression of expansion of gas within the same. When charring occurs about the electrode the result is to lessen or stop entirely the coagulating effect. These effects may easily be demonstrated by using the current on raw beef.

When the mucous membrane is coagulated, considerable hyperemia occurs about the area, accompanied by more or less edema.

Pain is a pronounced effect and varies in intensity in different localities. The mucosa of the nose is very sensitive. Coagulation in the posterior part of the nose is accompanied by a sensation of heat, and pain is experienced in the region of the distribution of the maxillary branch of the fifth nerve. These effects are present even after very thorough cocainization of the nasal mucosa.

The tonsil lymphoid tissue is rather slightly sensitive, and applications may be made in some instances without even a local anesthetic. On the other hand, the plica triangularis and the pillars are very sensitive.

Pain in the ear is experienced frequently when the current is applied near the capsule of the tonsil in the region of the upper pole and in the middle of the tonsillar fossa. This pain appears in spite of thorough surface application of 10 per cent cocain solution, but it is not unbearable.

COMPLICATIONS.

Serious secondary hemorrhage has been reported following coagulation of lesions in the vicinity of the larger blood vessels. Preliminary ligation of these vessels has been advised in such instances.

Other untoward results are usually due to faulty technic, such as: (1) Accidental contact of the active electrode with other parts than those treated. (2) Faulty contact of the indifferent electrode, resulting in burns. I have found no reports of other complications directly due to the nature of the method.

LESIONS TREATED BY THIS METHOD.

Electrocoagulation has been used with varied degrees of success and failure in the following conditions in the nose and throat.

Portman[11] reports excellent results from its use in chronic tuberculosis of the interior of the nose as well as lupus and atresia of the nares. He speaks of it as the method of choice in the treatment of membranous atresia of the posterior nares and nasal synechia. The atresia, however, has a tendency to return.

Dr. Dan Mackenzie advises diathermy—that is, electrocoagulation—as the method of choice in the treatment of lupus of the

interior of the nose, but believes radiation yields better results externally.

Sir St. Clair Thomson mentions diathermy as one of the palliative measures in the treatment of inoperable cancer of the nose and throat.

The use of these currents in cancer of the larynx is limited, according to Thomson, to epithelioma of the epiglottis and the postcricoid region, where it may be used as a palliative measure for the relief of pain.

New[7] reports 21 patients, comprising 30 per cent of his cases of malignant tumors of the antrum, living from one to eight years after combined treatment with radium and cautery. He says: "By the use of cautery in the form of soldering irons or diathermy and by the use of radium in the treatment of malignant tumors of the antrum, operative deaths are eliminated and more patients are cured than by previous operative methods."

Dr. Dan Mackenzie[8] reports favorable results where cutting and coagulating currents are used in treatment of cancer of the pharynx, tonsil and tongue. He regards "diathermy as one of the best palliatives in our possession at present," but says "it may profitably be associated with the use of radium and deep X-ray therapy."

Mackenzie gives the following reasons for its use: (1) It prevents the operator from squeezing living cancer cells into the lymphatic vessels draining the affected area.

(2) It prevents the sowing of the operative field with living cancer cells.

(3) It destroys the bacteria of sepsis in the tissue adjoining the field operation.

The use of electrocoagulation in the treatment of chronic tonsillitis has been reported upon by numerous authors, such as Portman and Moreau, Mackenzie, Bourgeois and Poyet, Hofoendahl and others. There is a concurrence of opinion that this method cannot replace the ordinary surgical methods of removal of tonsils.

Hofoendahl and others recommend the removal of tonsils by electrocoagulation in persons suffering from high blood pressure.

tuberculosis, anemia and in certain highly nervous patients who dread and refuse ordinary surgical procedures.

The method is particularly well adapted to the removal of small bits of tonsil tissue appearing in the tonsillar fossa after tonsillectomy and in the removal of masses of lymphoid tissue from the pharyngeal wall.

In the larynx, electrocoagulating currents have been used in the treatment of lupus and tuberculosis. Henri Bourgeois and Gaston Poyet,[9] however, stated that the galvanocautery yields better results in the treatment of tuberculosis of the larynx, for the reason that scar tissue is produced by the cautery.

Tobey of Boston[5] reports successful removal by electrocoagulation of two cysts of the anterior surface of the arytenoids after recurrence following the surgical removal. He also reports the successful removal of a varix of the vocal cord.

Hubbard and Galbraith,[10] on a series of cases, report the successful removal of papillomata of the larynx by fulguration and diathermy.

REMOVAL OF TONSILS—TECHNIC.

During the past year I have been using electrocoagulation in the treatment of chronic tonsillitis. Only such cases were treated as refused the regular surgical procedure. Our number, therefore, is small.

The tonsils are treated alternately, allowing at least a week to elapse between treatments. Thus, a two weeks' period is given each tonsil for recovery from the previous seance.

A 10 per cent cocain solution is used for anesthesia and is applied with a small cotton swab from which the excess of solution is wrung out. The mucous membrane of the tonsillar pillars, the tonsil and the area below the tonsil, as well as the base of the tongue, are gently massaged four or five times, at intervals of a minute or two, using the same applicator without dipping it again in the solution. Thus it will be seen that very little cocain is used.

The machine is set so that when short circuited a reading of 3,000 milliamperes is obtained when the current is turned on.

After being so set, the rheostat setting may be changed to reduce the current when necessary.

The active electrode consists of a heavy steel needle with its end bent so as to form a short hook which is tapered abruptly to a point.

The indifferent electrode is a sheet of block tin about six by ten inches in size. This is placed over the patient's back. In cold weather it should be warmed before being placed.

With pillar retractor and tongue depressor, the action of the active electrode should at all times be kept in view.

In applying the current to the tonsil, the needle point is inserted into the tonsil tissue for a distance of from one-eighth to one-quarter inch, keeping the point of the needle toward the middle of the tonsil. Stepping on the foot switch at this moment, one waits for the appearance of the white ring, when the current is immediately shut off and the needle withdrawn. This procedure is repeated until the surface of the tonsil has been covered with punctures about one-quarter inch apart.

This constitutes the entire procedure, which, of course, must be somewhat varied after the second and third treatment. At this time only a thin layer of tonsil tissue and capsule will have been left. One must not insert the needle into these thin layers of tissue, but simply lay the convex border of the curved needle against the tissue and apply the current only for an instant. In this latter instance, the rheostat setting must be changed so as to reduce the coagulating effect.

Care must be taken so as not to coagulate the mucous membrane covering the pillars, as this causes unnecessary destruction and postoperative pain.

In the patients I have so far treated, I have noted the following results and effects: After none of these treatments has there been sufficient pain to cause the patient to refrain from eating, and no patient complained of any feeling of illness.

Pain to a certain extent was experienced during all of these treatments, but was evidently so little as to cause but slight annoyance. Mingled as it was with subdued nervousness during the application, it was usually hard for the operator to know whether anxiety or pain was the greater.

Pain usually occurs during the two or three days following a a seance, but in about one-quarter of the instances no pain or discomfort was noticed at all. In a few instances pain occurred during the third and fourth day only and was felt in the homolateral ear as well as in the throat.

Pain in the ear on the side of application appears almost constantly when treating in the region of the capsule in the middle of the tonsillar fossa and the upper pole.

No bleeding occurred during any of the treatments aside from an occasional ooze from the prick of the electrode. This always stopped immediately with the application of the current.

Bleeding occurred twice in the same patient five days after coagulation. It occurred in the same tonsillar area but was not of a serious nature. On each of these occasions the patient was seen within a half hour after bleeding was first noticed by the patient and no active bleeding was then taking place. A small clot had formed in the tonsillar fossa on each occasion. This was removed and the fossa washed with warm normal saline solution. No further trouble was experienced although this same tonsil area was treated twice later.

At no time was there any evidence of toxemia following these treatments. The pallor which is sometimes seen after tonsillectomy was at no time present.

The tonsillar fossa in which the tonsils have been entirely removed, present a smooth, soft and pliable surface with no dense white scar tissue.

CONCLUSIONS.

That this is a time consuming method, both for the patient and physician, cannot be denied, since an average of eighteen treatments was given each patient.

On account of the small number of treatments given, no conclusions of any great value may be drawn, in so far as removal of tonsils is concerned. I am presenting the results of these treatments simply at their face value.

I wish to thank Dr. George Fetterolf for his helpful suggestions and much appreciated assistance in the preparation of this paper.

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LVIII.

THE SPHENOIDAL SINUS SYNDROME.*

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The symptom combination to which we desire to direct attention is exemplified in the case of a farmer, Mr. J. W., aged 67 years, who was referred to us with the following letter from his family physician:

"About a week ago, Mr. W. had a 'dizzy spell' while walking along the street. Had to sit down to recover from it. He has had a chronic hypertension, systolic blood pressure around 190 to 210.

"There seems to be a question of a cerebral lesion. He has developed a tinnitus in the left ear, with marked decrease of hearing. This morning he called and said he had become totally deaf. He has had a pain behind the left ear, but there seems to be no infection of the middle ear. It would not surprise me if the trouble went deeper than the labyrinth."

The patient, a farmer of rugged physique and constitution, stated that he had endured for several years a buzzing in his right ear, together with considerable loss of hearing in it. His left ear, however, had never troubled him, and he had gradually come to pay no attention to the other. There had been also slight attacks of dizziness for several years, sometimes of sufficient severity as to necessitate his sitting down a short time to avoid falling. Then, in the attack mentioned by his family doctor, he had acquired a severe humming noise in his left, or good ear, which was accompanied by marked loss of hearing and more frequent occurrence of dizziness. He was now annoyed by a pain in the back of his head on the left side. There was complaint of marked increase of vertigo from coughing or sneezing; and he thought that at these times he had some sensation of rotation. Generally, his dizziness consisted only of light headedness or a feeling of falling. There was no history of otorrhea or of pain in his ears. Aside from the present complaint his

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health had always been good, save for a blood pressure persistently high during the past few months.

Examination of the nose revealed a slight septal deviation to the right, with good airways, membranes not swollen or inflamed, and a drop or two of mucopus clinging about the region of posterior ethmoids and sphenoid on the left. The teeth were artificial. The tonsils were small, retracted and fibrous, not red; no pus was expressed. The uvula was slightly edematous and relaxed, but there was no asymmetry of action in the soft palate. The walls of the nasopharynx were wet and slightly thickened, but not red. The cervical lymphatic glands were not palpable, and the thyroid was not enlarged. Both ear drums showed normal light reflexes. Drums and ossicles moved in unison, and there were no signs of inflammation in either ear. Audiometric tests showed an average hearing loss of 43 per cent for each ear, with the maximum lowering in the tones above 1024 d. v. or C^4. Bone conduction was reduced 30 per cent. A Yankauer bougie easily traversed each eustachian tube. Inflation was not difficult, and notably gave no sensation of vertigo, despite the patient's statement that coughing and sneezing did so. Responses to vestibular examination were entirely normal, save for some uncertainty in the pelvic girdle movements. There was a fairly marked mitral systolic murmur present. The pulse rate was 72 per minute, and the temperature 97.8° F. The systolic blood pressure was 190 mm. mercury.

On lavage of the maxillary sinuses clear fluid returned from each. From the right sphenoidal sinus some granular débris and mucous shreds were recovered. Washing the left sphenoidal cavity released an accumulation, approximately two drams, of what appeared to be an intimate mixture of darkened blood clot and mucopus.

Upon the removal of this coagulum from his sinus, the man's expression of patient endurance suddenly underwent a change to one of amazed incredulity, as he exclaimed that his hearing had returned and that the noise was gone from his left ear.

This dramatic experience was repeated at each subsequent treatment, until the annoying symptoms became greatly ameliorated. The dizziness stopped entirely for a few days after each

washing and gradually ceased to appear as his sinus condition improved.

Interest in the combination of posterior pain, vertigo and tinnitus led us to watch carefully for its occurrence in later patients and to examine former case records for it. This study revealed that except in point of severity, we had seen many exactly similar cases and had considered these symptoms as being merely a part of the general sinus symptomatology. In many subsequent patients, in whom the combination appeared, we found it possible regularly to afford them relief by lavage of the sphenoid sinuses.

Hearing loss, so prominent in the patient described, is not at all a constant finding, only occasionally appearing in sufficient degree to be noticeable. The triad, however, seems a definite combination appearing sufficiently often to be of diagnostic significance in chronic or subacute sphenoid sinus involvement. Each phase of the group shows some points of interest which we have not, in a limited survey of the literature, found mentioned.

Occipital pain occurs frequently with infection of other sinuses, as well as in brain tumor, ocular disturbances and some general conditions; but it is not identical with the type here referred to. We have not found this particular discomfort in any condition where sphenoid sinuses were not diseased, as demonstrated by lavage of mucus or mucopus from them. The differentiating point is one of location. Ordinarily, occipital pain seems to center in the suboccipital region—that is, about where head and neck join—and may affect one side, both sides, or be exactly in the midline. Such distress may and frequently does arise from trouble in the posterior sinuses. Characteristic sphenoid pain is often written of as being in the mastoid process, a description which we have not been able to verify. In our observation, this painful sensation appears to center at a point midway within and .5 cm. to 1.5 cm. above a line drawn from the external auditory meatus to the tip of the external occipital protuberance. This is, of course, somewhat behind the mastoid process and not in anatomic or physiologic association with it.

Sphenoid discomfort varies from a heavy, steady aching to lancinating, stabbing pains, or a burning sensation; and the area described above is often sensitive to superficial pressure.

Anatomic observations seem to identify the superior cervical sympathetic ganglion as the switchboard through which this particular pain reference is accomplished. Its superior branches help to form the carotid plexus. From this plexus arises the great deep petrosal nerve, which anastomoses with the great superficial petrosal (from the facial at geniculate ganglion) to form the vidian nerve. The superior cervical ganglion also communicates with the four upper cervical nerves, through its gray rami communicantes. This serves to connect each vidian nerve, which runs along the floor of its corresponding sphenoid sinus, with the occipital nerves, major, minor and tertius, arising from the three upper cervical spinal nerves. The three occipital nerves, through liberal anastomoses of their sensory skin branches, supply an area to which vidian nerve disturbances may be referred.

Intrasphenoid tissue seems endowed with a high degree of sensitiveness to any inflammatory change; and it is surprising how the removal of a small kernel of mucopus, by lavage, will give marked relief of symptoms. The word "kernel" is used descriptively, since these minute collections appear as little balls or globular masses, possibly so formed by rolling about from one part of the sinus to another. Theoretically, one might thus partially explain the observation that sphenoid pain and dizziness are so often intensified by movement of the head. Pain from these sinuses is frequently complained of as being worse when the patient reclines, and is at least a little relieved by lying face downward or turning the face partly down over the end of a firm pillow.

A fact often overlooked, and which was long ago pointed out by Sluder, is that free opening of the sphenoid sinuses, even where the windows are ample and permanent, does not prevent secretion from collecting in them and causing pain. The very proclivity of these cavities to invade pterygoid processes and advance so far downward, almost at right angles to the direction of their drainage, makes it extremely improbable that the head will ever be kept long enough in a position favoring their unloading to offset the tendency toward collection of slowly produced secretions, as it were, at the bottom of the well. Failing to recall this, we may be led to exclude them as a possible seat of severe

symptoms, when warm lavage of their interiors would afford both relief and indisputable evidence of their culpability.

Occasionally pain from a sphenoid sinusitis is localized in the ear itself. This may come through the sphenopalatine ganglion. However, experience seems to bear out that most so-called neuroses of this region may be more effectually relieved by proper attention to sphenoid, maxillary and ethmoid sinuses than by injection or other treatment of the ganglion itself.

Head noises are quite commonly an accompaniment of sphenoid sinus disease. In our experience, they are most frequently described as of a humming or buzzing character. In at least a few instances the patient cannot seem to tell which ear is noisy and places the tinnitus as being "in the center of the head."

Sluder, Schaefer and others have demonstrated the close relationship between the eustachian tube and a sphenoid sinus which is prolonged down into the pterygoid process, reaching to the bifurcation of its plates and even beyond. That secondary congestion of the otopharyngeal tube from this intimate contiguity might produce a tinnitus is undeniable. But might it not also reasonably arise as a malevolent product of sensory nerve reflex mechanism? The following quotations are suggestive.

Gleason[1] remarks: "The manner in which disease of the digestive tract, teeth or nose produces tinnitus is through the nervous connection, more or less direct, with the inferior cervical sympathetic ganglion, which supplies the nervi vasorum to the occipital artery and its branch, the internal auricular. Irritation of the inferior cervical sympathetic ganglion would cause tinnitus as the result of dilatation of the arterioles of the cochlea, which at first pulsating, would afterwards become constant in character as the result of trophic changes resulting from increased blood supply."

Barnhill[2] says: "The actual cause of tinnitus lies in the hypersensitive nerve endings of the labyrinth. For, whether the cause of head noise is in the blood stream, nervous system or conducting portions of the ear, it seems reasonable to suppose that the terminal portions of the acoustic nerve must be abnormally irritated before any sound other than normal can be perceived."

Undoubtedly, many factors are operative in the production of tinnitus, and one can scarcely accept the presence of any special changes in cochlear tissues as producing head noises which completely disappear within a few seconds after lavage of a sphenoid sinus. More conceivably might such a type be the locus of expression actuated by some disease area or focus, such as the sphenoid sinus.

Disturbance of orientation in disease of the sinuses can scarcely, as a rule, be dignified by the name of vertigo. Yet it does, at times, assume an actual rotatory quality. Hunt[3] states that "dizziness is a mild form of vertigo," while others have written that any study of vertigo is essentially an ear study. Actually one is led to speculate as to the possibility that some agency quite removed from the vestibular apparatus may occasion dizziness or giddiness, until its agitation rises above the stimulation threshold necessary for its extension to the labyrinth, with the consequent production of a sensation of motion or rotation. Certainly, if dizziness from the sphenoid sinuses is produced through disturbance of the internal ear, that organ must assume in the nerve hookup a place corresponding to that assumed by the spot behind the mastoid in the reference of pain from the sphenoids. In view of the ready relief obtained from ridding an affected cavity of its abnormal secretions, one can scarcely accept any theory of toxic effect upon the vestibular apparatus as explanatory of the phenomenon. Sluder[4] reported a case in which genuine rotatory vertigo, apparently from posterior sinus disease, was relieved at once by cocainization of the sphenopalatine ganglion. Our patient, already referred to, complained of rotatory vertigo only when he sneezed or coughed; but he had frequent brief periods of dizziness, which ceased bothering him for so long as the sinuses were kept free of abnormal secretions. One person suffering from the dizziness of sinus disease attempted to describe it by referring to the sensation experienced in his hand from striking the ulnar nerve, in its groove between the olecranon and the medial humeral epicondyle. Of course, it goes without saying that this symptom is itself not limited to disturbance of sphenoid cavities, but frequently results from disease in the other sinuses as well.

Giddiness of subacute or chronic sphenoid disease is often complained of as "light-headedness" or a "feeling of falling." It is usually experienced upon motion of the head, as when walking, stooping, rising or turning; and we might offer for consideration possible insult to inflamed and already resentful vidian nerves—in their almost unprotected position upon the sphenoid floor—by even slight pressure of contained secretions moving over them. It seems within the range of possibility that so little disturbance might initiate an explosion of nervous energy in an inflamed nerve, which even in health is so extremely sensitive as is the vidian.

Whatever its mechanism of production, this light-headedness constitutes a source of great annoyance and worry to the patients. In some it even becomes disabling.

Whether the combination of posterior pain, tinnitus and dizziness is invariably present in disease of the sphenoid sinuses cannot at present be fairly stated. However, we have found it so frequently, in patients having this trouble, that we believe any individual complaining of these symptoms should have, among other things, a careful investigation of the sphenoid cavities. Naturally the comparative severity of the symptoms may vary greatly in different patients, and one member of the triad may be absent. However, we believe they occur in combination with sufficient regularily to justify the tentative designation of sphenoid sinus syndrome.

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LIX.

THE SIMPLE MASTOID OPERATION: A MODIFIED TECHNIC.*

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The purpose of this paper is to describe an operative technic which is based upon anatomic studies from the sectioning of more than eighty temporal bones, and also to overcome certain marked disadvantages of the classical simple mastoidectomy.

A. ANATOMIC CONSIDERATIONS.

The mastoid sinus is surgically divided into five cell groups, namely: (1) The retrofacial cells, (2) the tip cells, (3) the perisinus cells, (4) the petrosal angle cells, (5) the zygomatic cells. It is with the zygomatic group that these anatomic considerations are concerned. The zygomatic cells lie in the posterior root of the zygomatic process of the temporal bone. These are seen to be divided into two groups, a posterior and an anterior group. The posterior group is wedge shaped, with the apex pointing forwards. This is the group that is removed in mastoid operations, and which is well known to otologic surgeons and needs no further description. It is the anterior group that is not exenterated in mastoid surgery.

On the external surface of the temporal bone, this anterior group is triangular in shape, its base is marked superiorly by the anterior extremity of the linea temporalis, inferiorly and posteriorly by the anterosuperior margin of the meatus of the external auditory canal, and inferiorly and anteriorly by the posterosuperior margin of the glenoid fossa. The inferior tip of this triangle is marked by the postglenoid process. The shape of this anterior group of zygomatic cells is a triangular truncated pyramid, the base of which has just been described. It

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extends medially and slightly posteriorly to terminate as the anterior third of the lateral wall of the epitympanic space. The posterior margin of the pyramid communicates directly with the anterior margin of the posterior zygomatic group. This junction of both zygomatic groups forms a very narrow space, and, on this account, the anterior group is readily overlooked in mastoid surgery.

The anterior zygomatic group of cells is accessible surgically by lowering the superior wall of the auditory canal down to a variable distance, about one-third of its depth, and removing it as far forward as the anterior canal wall. The cells are now accessible to the gouge or curette.

It is just as important to remove these cells as it is to remove the tip cells, for the anterior zygomatic group are just as liable to be focal centers as are other remote corners of the mastoid cellular structure. I have seen persistent otorrhea following mastoidectomies clear up after reopening the mastoid and exenterating more of the zygomatic cells. It is very likely that the failure to expose these cells is the cause of large numbers of persistent unaccounted for otorrheas following the simple mastoid operation.

Occasionally, in the operating room, one meets with what are called extensive zygomatic cells—i. e., cells that seem to reach further forward than those usually met with. This apparent forward extensiveness is due to the greater cellular structure between the dural plate and the superior canal wall—that is, where the apices of the posterior and anterior groups meet. If the apices are narrow, their junction is interpreted as being the limit of the forward extension of the zygomatic cells, and where the apices are broad it is interpreted as being extensive zygomatic cells.

B. SURGICAL CONSIDERATIONS.

The chief advantage of the classical simple mastoidectomy is in the large exposure and view obtained by the removal of the entire mastoid roof or cortex. The purpose of the technic about to be described is to utilize the advantage of a large exposure and to remove the disadvantages of the usual simple mastoid, together with the exenteration of the anterior group of zygomatic cells already described. The disadvantages alluded to above are:

1. The exenterated mastoid cavity cannot be visualized during the postoperative treatment.

2. Drainage and after-treatment of the mastoid cavity are carried out through the postauricular opening which has been left for that purpose and which has distinct disadvantages, namely:

(a) The presence of a persistent postauricular fistula may result.

(b) The primary mastoid incision is left to unite either throughout its entire length or at its inferior segment, by secondary intention, which results in a prominent scar.

(c) It necessitates the wearing of a head bandage for long periods owing to the postauricular drainage. The head bandage is very uncomfortable and is usually an economic loss to the patient by detaining him from his gainful occupation.

(d) In case of recurring abscess of the middle ear or antrum, it is necessary to provide for drainage via a postauricular incision. The disadvantage of this, other than an ugly discharge in such a conspicuous place, is the distance through which the pus must go in order to escape from its source—the middle ear or the antrum—to the exterior.

3. The periosteum of the mastoid cortex, especially of the posterior segment, is greatly traumatized, and therefore seldom brought well together in closing the mastoid incision.

It is aimed to overcome the above mentioned disadvantages by closing the entire postauricular mastoid incision to allow of primary union; and in providing for drainage and after-treatment of the mastoid cavity through the external auditory canal by cutting a window in the membranous lining of the posterior auditory canal wall after the posterior bony wall had been lowered; and by elevating the periosteum intact from the cortex, via an incision in the canal wall. And in addition, the anterior group of zygomatic cells which have been described above are exenterated.

Some of these advantages are overcome in the radical operation, since here the posterior auricular incision is closed completely, and drainage and after-treatment are carried out through the external auditory canal.

THE OPERATIVE TECHNIC.

Exposing the Mastoid Cortex.—The initial incision is made in the fashion to be designated for the purpose of elevating the periosteum from the underlying bone to minimize the trauma to this soft tissue. This incision is made in the posterior membranous canal wall at the junction of the membranous with the cartilaginous meatus. The place of the incision is best located by palpating with the index finger for the junction of the canal wall with the planum mastoideum. The auricle is drawn taut by pulling it upward and backward. The incision is carried from the superior to the inferior canal wall. A Freer elevator is inserted into the incision and, getting a cleavage between the periosteum and the mastoid cortex, the periosteum is elevated from the bony mastoid roof by lateral, side to side motions of the Freer elevator and at the same time advancing it to uncover the greater part of the mastoid cortex. A cotton tampon inserted into the meatus will control bleeding.

This technic is identical with that employed in submucous, nasal septal resections, where the mucoperichondrium and mucoperiosteum are being elevated. The advantage of this procedure is that the periosteum is not traumatized as much as when it is separated following the retroauricular incision.

The retroauricular mastoid incision is next done in the usual way, care being taken, however, to keep the incision within one-quarter inch of the posterior attachment of the auricle and to bring the upper end well forward, in order that the auricle can be drawn sufficiently forward to permit of the separation of the membranous lining of the posterior and superior bony canal walls down to the annulus tympanicus.

The Bone Operation.—The mastoid cortex is shaved off with a gouge, extending from the linea temporalis above to the mastoid tip below. The retrofacial cells and some of the cells in Trautman's triangle are now removed with a curette. The posterior canal wall is now lowered as in the conservative radical operation—i. e., from its inferior limit it is shaved off, sloping gradually toward its junction with the superior canal

wall, and extending medially to within one-third of its depth. The remaining mastoid cells are next curetted away.

Exenteration of the Zygomatic Cell Groups.—The superior wall is removed forward to the anterior canal wall and, in order to gain sufficient access to the anterior zygomatic group, a chip of the superior external part of the anterior canal wall is removed. These two maneuvers lay open both zygomatic cell groups, which can now be exenterated either with the chisel or curette. This completes the bone operation.

Operation on the Soft Parts.—In this step a window is cut into the membranocartilaginous portion of the posterior canal wall to correspond in size and position with the opening in the bony posterior canal wall. The purpose of this window is to allow of communication between the mastoid cavity and the external auditory canal, which provides a route for drainage and after-treatment of the mastoid cavity. Plastic flaps are omitted entirely.

The mastoid cavity is now lightly packed with gauze strip, the distal end of which is brought into the external auditory canal through the window cut into the membranous lining. A second gauze strip is inserted into the external canal close to the membrana tympanum. The retroauricular mastoid incision is closed completely, as it is not necessary to leave an opening for drainage and packing, this having been provided for via the external auditory canal. The usual mastoid head bandage is applied and is omitted after the first dressing. This completes the simple mastoid operation.

SUMMARY.

The advantages claimed for this operative technic are:

1. In the removal of the anterior zygomatic cell group which communicates directly with the remainder of the mastoid cells and with the epitympanic space, and is therefore subject to pathologic involvement in otitic diseases.

2. Far better preservation of the periosteal lining of the mastoid cortex, and this provides for better healing of the operative wound.

3. Complete closure of the retroauricular mastoid incision allows of primary union and minimal scar formation and disfigurement.

4. Drainage and packing of the mastoid cavity via the external auditory canal forestalls possible persistent retroauricular fistula formation, and allows of visual examination of the mastoid cavity throughout the healing process, the advantages of which are obvious.

5. In case of recurring abscess, it can be drained by an incision through the posterior wall of the auditory canal. Drainage via this route is more desirable than via the mastoid roof, because firstly, the abscess is nearer to the internal part of the auditory canal, and therefore has less distance to travel in draining, and secondly it is far better from a cosmetic viewpoint.

6. The head bandage can be omitted after the first dressing; the advantage of this has already been mentioned.

7. The exenterated mastoid cavity can be visualized during the postoperative treatment by inserting a speculum into the cavity via the external auditory meatus. The advantages of this are obvious.

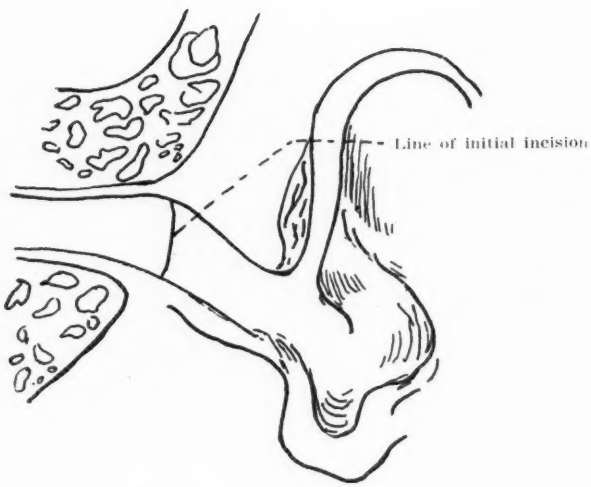

Fig. 1. Posterior wall of the cartilagenous and osseus meatus.

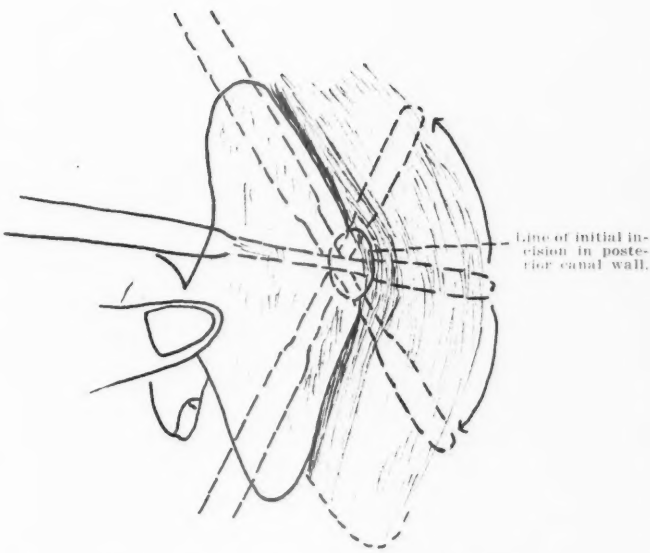

Fig. 2. Showing method of elevating periosteum.

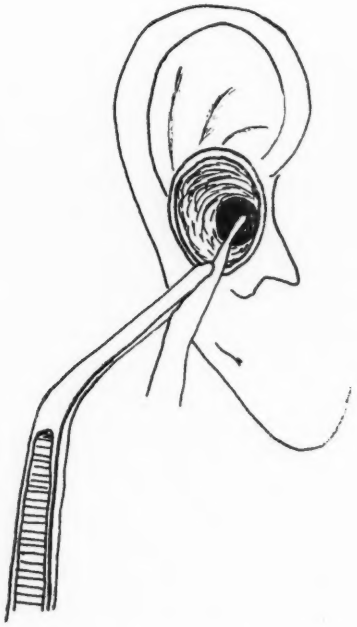

Fig. 3. Showing packing of mastoid cavity.

LX.

A STUDY IN OTOSCLEROSIS.*

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The purpose of this paper is to present the results of a clinical study of 1,000 cases of deafness, to note the relative frequence of the occurrence of otosclerosis, and to ascertain, if possible, from the ideas of otologists, past and present, just what this condition may be.

How long this disease has been in existence is unknown. The biologic origin of the acoustic apparatus undoubtedly dates from the amphibian, and whether some faulty anlage has continued to follow its development through the stages of evolution or whether it is a decadent condition in man, since the apex of auditory acuity is found in wild animals, is debatable.

The history of otosclerosis up to the time of Toynbee, in 1851, is a collection of fragmentary bits of anatomy and physiology, some correct and some incorrect, but all leading to a basic knowledge essential to later studies. Toynbee examined about twelve hundred cases of diseased ears anatomically, and became interested in stapes ankyloses, which he attributed to a "rheumatoid arthritis." His description of these conditions, with their varying degrees of ankyloses, is correct today. His study of signs and symptoms and his observations of pathologic findings are exactly ours, but he did not find or even suspect that many of his ankylosed stapes were produced by labyrinthine lesions. Von Tröltsch, in 1862, first used the term "otosclerosis," but, according to his description, it was a chronic catarrhal otitis media and not that progressive deafness which develops in the presence of a normal drum and tube, and in which we find lesions of the labyrinthine capsule. In 1867, Moos gave reasons for consider-

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ing otosclerosis a primary disease of the labyrinth, and from then on intense studies were made by the foremost otologists of all countries. Politzer was one of the first to report findings and opinions, and valuable contributions were made by Bezold, Siebenmann, Brühl, Alexander, Manasse, Gray and others, all supporting the labyrinthine theory.

Agreement concerning a localization of the lesion, however, did not settle controversies concerning a conception as to just what it is, what causes it, or what its exact pathology may be. These questions are still with us, and when I present 1000 cases of deafness, classified to show the percentage of otosclerosis present, we must recognize these differences of opinion, and by considering each other's interpretations bring about a better understanding of the term. Ten otologists representing, I believe, all the different schools of interpretation, show by their definitions the following points of agreement:

1. A progressive deafness.
2. Normal drumheads and eustachian tubes.
3. Lesions in the labyrinthine capsule.

They also show the following points of disagreement:

1. Essential nature of stapes ankylosis.
2. Differences in etiology.
3. Differences in explanations of pathology.

The best definitions today do not attempt to define in terms of etiology and pathology but rather in terms of clinical findings and such pathologic observations as can be agreed upon by all. Joseph, in 1920, defined otosclerosis as "A progressive hardness of hearing without visible changes in the tympanic membrane or tube, the pathologic basis of which is a peculiar focal disease of the bony labyrinthine capsule, that secondarily leads to stapes ankylosis and sometimes to degeneration of the labyrinth." Most of us can concur in this definition and still differ on points of etiology and pathology.

At the present time there are seven main theories as to the etiology of otosclerosis, and all have the support of able men.

1. The infectious theory has had little support in recent years, although Kerrison and Lake have advised the removal of all foci of infection for empirical reasons.

2. The toxic theory has been considered from various points of view. A gastrointestinal autointoxication, a rheumatic or gouty diathesis, a uric acid diathesis and similar ideas have been advanced.

3. The deficiency theory reduces itself merely to the question of vitamins and naturally calls forth analogies to rickets, osteomalacia, arthritis deformans and similar conditions.

4. The Endocrine Theory.—The incidence of deafness and latent tetany has been noted for some time. That an altered gland secretion is responsible in some way for this disease has been observed by Pollock, Stevenson and others.

5. Heredity, we all agree, is the one constant factor in its etiology.

6. The circulatory stagnation theory states that otosclerosis may be the result of the failure of a local blood supply which terminates in the absorption of normal bone and cartilage, the formation of a spongy bone, as might be expected in areas where the blood supply has failed, and later a deposition of lime salts such as occurs in any such area.

7. The neoplastic theory assumes that because of a faulty anlage, with or without an exciting cause, lesions which are essentially new growths develop in a highly complex embryologic portion of the labyrinthine capsule.

The pathologic controversies involved in this study usually pivot about unproved etiologic explanations of the conditions found or rotate around what should or should not, in the mind of a particular individual, be included in its definition and symptomatology. The essential features of the pathologic picture are observed and conceded by all; only details are controversial. That stapedial ankylosis is common in this condition is conceded; that it is necessary to it is denied; that the pathologic site is internal to the niche of the oval window and may spread over the promontory to the round window, or even occur sporadically throughout the cochlea or petrous bone, must be admitted. All apparently agree that a lesion, undefined at present, occurs in the labyrinthine capsule, which in the presence of an ear otherwise normal will produce signs and symptoms characteristic of otoscle-

rosis. Clinically we usually diagnose this condition when the stapes begins to ankylose; pathologically, it may be observed earlier.

What, then, is the picture that shows microscopically in the labyrinthine capsule? With the possible exception of those who still hold that the stapedial joint may be ankylosed after the form of an infective arthritis, and who therefore do not really support the labyrinthine theory, all seem to feel that the pathologic lesion, whatever it is, develops either within or in areas adjacent to the blood vessels. The early observers noted areas of bone absorption around the Haversian canals and other areas of new bone formation. To the absorption areas the term "spongification" was applied; to the new bone formation the term "recalcification." It is assumed that the spongification came first and the recalcification came afterward. Politzer, Hartmann, Katz, Brühl, Gray and others agree that this is essentially the picture. Alexander went further in his observations and noted an engorgement of the blood vessels and a marked increased cell formation, which apparently preceded the deposition of lime salts. This so impressed him that he called the condition "osteitis vasculosa." Manasse also saw the engorgement of the blood vessels and observed projecting into the lumen "sieve-like mantles" of osteoid and granulation tissue, which destroyed the labyrinthine capsule by simple displacement. More recently Gray, in discussing this, feels that the engorgement of the vessels results in a stagnation of the blood flow, the formation of thrombi, and the resultant bone absorption and recalcification that might occur in any area where the blood supply had failed.

Those that support the neoplastic idea concur essentially in the observations made, but differ, of course, in their ideas about the etiology and character of the lesion. They feel that the end lesions are a result of a progressive rather than a retrograde change, and that cartilage islands precede new bone formation, and that the whole process occurs in areas of highly complex embryologic development in which new growths are prone to occur.

So far we have discussed some of the historical, etiologic and pathologic aspects of otosclerosis. We shall now consider it clinically. In the diagnosis of this condition the following points

should be observed in each patient, and whether or not your diagnosis is otosclerosis depends entirely upon your interpretation of their importance and how they arrange themselves into a clinical picture which, in your mind, is otosclerosis:

1. *Progressive deafness.
2. Family history.
3. *Bezold's triad.
   (a) Raising of the low tones.
   (b) Increased bone conduction.
   (c) Negative Rinne.
4. *Normal middle ears and eustachian tubes.
5. Onset.
   (a) Puberty.
   (b) Pregnancy.
   (c) Menopause.
6. Bilateral aspect.
7. Tinnitus.
8. Paracusis willisii.
9. Promontorial blush.
10. Diminution of the tickle reflex, absence of wax, etc.
11. Dizziness, vertigo, disturbance of equilibrium.

Other points have been suggested and may be observed to determine their importance. Such are:

1. Beck's areas of rarefaction shown in the X-ray.
2. Drury's contraction of the visual fields.
3. Fatigue reactions noted in functional hearing test.

The stars represent those points without which, in my opinion, no diagnosis of otosclerosis can scientifically be made. The first point is conceded by all. The other two raise two points of controversy. First, Bezold's triad always presupposes a stapes ankylosis, and therefore eliminates the possibility of a clinical diagnosis of an otosclerosis of type 3 as described by Shambaugh. He, however, admits that this type cannot be clinically differentiated from a pure nerve deafness, and therefore this point ought not to be controversial. The second point (i. e., the third star) requires the presence of a normal middle ear and tube, which automatically eliminates all cases of otosclerosis which are said

to be complications of other forms of deafness. It is not my purpose to suggest that such complications cannot exist, but to state that such a diagnosis cannot be made clinically and that in all probability, if such a picture is present, the supposition is that it is not otosclerosis at all.

To complete this study, 1,000 cases of deafness have been examined and classified. The general classification and grouping is tabulated as follows:

Total number of cases examined	1000
Deafness resulting from:	
1. Chronic catarrhal otitis media (uncomplicated)	556
2. Chronic catarrhal otitis media (degenerating to nerve deafness)	114
3. Chronic catarrhal otitis media (degenerating to simulate otosclerosis)	26
4. Chronic purulent otitis media	60
5. Chronic purulent otitis media (degenerating to nerve deafness)	1
6. Chronic purulent otitis media (degenerating to simulate otosclerosis)	21
7. Chronic tubal catarrh (drum normal, all ossicles free)	43
8. Atresia of the external canal	2
9. Malingerer	1
10. Otosclerosis (with stapes ankylosis)	57
11. Otosclerosis (with stapes ankylosis and nerve degeneration)	7
12. Nerve deafness	93
Undiagnosed cases	19

The major forms of deafness thus represented, in terms of percentages would be as follows:

1. Middle ear deafness	77%
2. Uncomplicated tubal deafness	4%
3. Otosclerosis (with stapes ankylosis)	5%
4. Otosclerosis (with stapes ankylosis and nerve deafness)	0.7%
5. Nerve deafness	9%

There were 57 cases of otosclerosis with a stapedial ankylosis; seven others that had definite stapedial ankylosis and developed a marked diminution in bone conduction during the years in which they had been observed; and 93 cases of uncomplicated nerve deafness. Twelve of the latter gave histories suggestive of otosclerosis.

From the 64 cases of definitely uncomplicated otosclerosis, then, we tabulate the following:

Males	47%
Females	53%
1. Progressive deafness	100%
2. Family history	48%
3. Bezold's triad	100%
4. Normal middle ears and eustachian tubes	100%
5. Onset (puberty, pregnancy, menopause)	65%
6. Bilateral aspect	94%
7. Tinnitus	76%
8. Paracusis willisii	31%
9. Promontorial blush (not recorded)	
10. Diminution of the tickle reflex	86%
11. Dizziness, vertigo, etc.	4%

Most of these cases have been observed from two to ten years, and in several instances the diagnosis was delayed purposely to insure its correctness. The treatment varied along empirical lines, and will be further considered in another article. Audiograms were made in many of these cases and were inconstant in character.

DISCUSSION.

Kerrison discusses as a separate topic, "Otosclerosis Complicated by Chronic Catarrhal Otitis Media." Does the existence of the one preclude the existence of the other? It is possible, perhaps, to settle this question on postmortem examination, but a fine histopathologic differentiation must be made between areas of otosclerotic spongification and areas of atrophy that have resulted either directly from a stapedial ankylosis or indirectly from a complicating vascular disarrangement secondary to the ankylosis.

In this series there were twenty-six cases of chronic catarrhal otitis media which over a period of years have assumed many characteristics of an otosclerosis. These, in the past, presented signs and symptoms of eustachian closure, drum retraction and ossicular rigidity in various forms and degrees, and were treated by inflation and otomassage. Years after, many of them developed an unusually permeable tube, a dry, scaly external auditory canal and a drum which remained either slightly retracted or normal. In certain cases the skin of the auricle seemed to lose some of its normal firmness. The probable explanation of this is

that, as a result of a chronic catarrhal otitis media which had produced a long continued congestion within the tympanum and an ankylosis of the stapes, secondary atrophic changes took place in the petrous bone and its contents so that the adjacent nerves, especially the fifth, seventh, eighth and ninth, are affected. The result is a mild atrophy of their corresponding peripheral regions. As a result of this, the tubes open, the external auditory canal becomes dry, and the extrinsic muscles of the ear lose their normal tonus. If the original lesions were not in the cochlea, or simultaneously in the cochlea and the middle ear, how does one account for the peripheral atrophic changes following secondarily upon, and the middle ear pathology changing after atrophic changes in the petrous bone? These do not appear to me to be cases of otosclerosis complicated by chronic catarrhal otitis media but forms which have degenerated into a similar condition.

Chronic purulent otitis media can obviously produce a like chain of pathologic events with all their resulting signs and symptoms. Of the 82 cases of this type, 21 degenerated into forms that could easily be called otosclerosis.

CONCLUSIONS.

1. In a series of 1,000 cases of deafness, less than 6 per cent were definitely diagnosed as otosclerosis.

2. The variable signs and symptoms of otosclerosis occur in the following relative frequency:

1. Family History	48%
2. Onset (puberty, pregnancy, menopause)	65%
3. Tinnitus	76%
4. Paracusis willisii	31%
5. Diminution of tickle reflex	86%
6. Dizziness, vertigo, etc.	4%

3. A careful histopathologic distinction must be made between a primary otosclerosis and secondary atrophic lesions produced by stapes ankylosis of middle ear origin.

4. Chronic catarrhal otitis media and chronic suppurative otitis media may degenerate into forms which simulate otosclerosis but clinically cannot be considered complications of it.

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LXI.

A COMPARATIVE STUDY OF THE BACTERIAL FLORA OF CLINICALLY NORMAL NASAL SINUSES.

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St. Louis.

There has been a large amount of work done on the bacteriology of the diseased nasal cavities and the accessory sinuses, but that dealing specifically with normal sinuses is surprisingly meager. There are only a few works worthy of mention.

Frans Torne[1] in 1904 made a very comprehensive study of the normal sinuses and the factors affecting the conditions observed. He examined fifty-two fresh cadavers, sixteen of which showed pathologic changes in the sinuses and were excluded. Autopsy was performed upon the remaining thirty-six within three quarters to twenty-five hours and most of them within six to eleven hours after death. The frontal and maxillary sinuses were entered with precautions for sterility and inoculations were made into plain broth, inactivated ascitic fluid, serum bouillon agar, glycerine agar, brain agar, and blood agar. He found twenty-nine with completely sterile maxillary and frontal sinuses. In one subject there were bacteria in both frontal, two in both maxillary, three in one maxillary, and one in both maxillary and both frontal sinuses. Of the remaining sinuses, about one-half were sterile. The sinuses were found to be sterile in all cases in which the examination was made within two hours and twenty minutes following death. The conclusion is drawn from these results that the frontal and maxillary sinuses in man are usually free from bacteria and that the entrance of bacteria takes place a few hours after death, the exact period varying considerably in some cases.

Nine out of seventy-two maxillary sinuses, and four out of sixty-eight frontals, showed the presence of bacteria, and the

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author believes, since others have found bacteria to be absent from the neighborhood of the ostium, that they are borne in toward the sinuses on foreign particles. The occurrence of the same type of bacteria in the maxillary and the frontal sinuses on the same side is pointed to as possible evidence for this theory. The organisms found were staphylococcus aureus, four times; bacterium coli, five times; streptococcus pyogenes, three times; staphylococcus albus, once; and a diphtheroid once.

Torne also found the frontal and maxillary sinuses of six freshly slaughtered calves to be sterile. Lampblack was sprinkled on the ciliated epithelium of the maxillary sinus of the calf and found to move toward the ostium at a rate of about 0.5 cm per minute. A similar experiment was performed on a human cadaver, dead seventeen to eighteen minutes, with similar results.

Calamida and Bertarelli,[2] 1902, examined the frontal and maxillary sinuses of twenty dogs and twelve human cadavers. The method of entering the frontal sinuses consisted of opening the skin and the periosteum with sterile instruments and making a hole in the sinus wall by means of a sterile scalpel. The mucous membrane lining the cavity was punctured with a glowing needle and a culture made on sterile bouillon or glycerine agar. The exact method of entering the maxillary sinuses is not given. All of the frontal sinuses except two which contained b. coli and b. proteus, respectively, were found to be sterile. In sixteen cases the maxillary sinuses were free from bacteria, while two showed "coccus auranticus" (staphylococcus aureus) and one each of "micrococcus albus" and staphylococcus albus. None of the twelve human subjects examined had died of infectious disease. They were examined within a few hours after death, none more than twenty hours afterward, but the method of entering the sinuses is not described. With the exception of one frontal sinus which contained staphylococcus albus all of the sinuses were sterile.

In order to test the resistance of the sinuses to the entrance of bacteria, an experiment was performed by these authors, in which easily recognizable bacteria were sprayed into the nasal cavities of dogs, and from four to twenty-four hours later the

frontal and the maxillary sinuses were examined for their presence. Ten dogs were given serratia marcessens, six bacillus subtilis and two pseudomonas pyocyaneus, using one to two cc. of a bouillon culture, and the dogs killed for examination eight to twenty-four hours later. Of those given serratia marcessens, the organism was recovered from the maxillary sinus five times, from the frontal three times and from the middle ear twice. Bacillus subtilis was recovered from the maxillary sinus twice and from the frontal once, while the sinuses of the two which were infected with pyocyaneous remained sterile. The positive findings were in animals killed eight to ten hours after the injection, while those killed four to six hours or eighteen to twenty hours afterward, were sterile with a single exception.

From these results it is apparently easiest for microorganisms to gain entrance to the maxillary sinuses, more difficult for the frontals and still more difficult to reach the middle ear. These differences are explained by the authors on an anatomic basis.

An examination of the sinuses of fifty cadavers, by E. Frankel (1896),[3] showed twenty-eight of them not diseased ("completely intact"), and of these thirteen were sterile. Of the remainder, four were not examined bacteriologically and the others contained one to several kinds of bacteria. The pneumococcus (diplococcus lanceolatus, Frankel) was the principal organism found. Eleven maxillary, six frontal and five sphenoid sinuses contained bacteria. The author believes that bacteria occurring in normal sinuses play an important rôle in inflammatory processes of the respiratory system.

The sinuses of twenty-nine persons, including doctors, nurses and nondiseased persons working about a tuberculosis sanitarium were examined for the tubercle bacillus by Strauss[5] (1903). The material was washed out on a sterile cotton tampon and guinea pigs injected. Nine animals injected with the washings from two doctors, six nurses and one nontuberculous worker developed tuberculosis. It was concluded that infection in the case of this disease comes through inhalation.

It is evident since the mucous membrane in the upper nasal passages is continuous with that of the nasal sinuses that the

bacteriologic condition of the former is of importance in this connection. A considerable amount of work was done in the early days of bacteriology on the bacteriology of the normal nose. In many cases the methods used were entirely inadequate. Park and Wright (1897) examined the secretions from the noses of ten healthy persons. Bacteria were found in all of them, but as admitted later, the method of collecting was not such as to exclude the possibility of contamination from the vestibule, which is always rich in microorganisms. In a later work[7] the vestibule was sterilized with a 1/2,000 solution of mercury chloride and the nasal mucus collected on a swab. In sixteen cases out of thirty no growth was secured. They conclude that the comparative scantiness of microorganisms is due to the combined action of gravity and cilia, to the fact that the nasal mucus is not a good medium for the growth of bacteria, to the filtering action of the vibrissæ and to the fact that the inspired air ordinarily contains few pathogenic germs.

Von Besser[15] (1889) used the method of introducing a platinum loop, three to four cm., into the nasal cavity and spreading the small amount of material secured on a plate. He found that the bacteria present ranged from a very few to countless. It is evident that the scantiness of the material and the danger of contamination from the vestibule would render such work unreliable.

Wurtz and Lermoyez[9] (1893) concluded that if one takes proper care to reject the first drops of nasal mucus obtained by excitation of the pituitary membrane it does not generally contain microorganisms and possesses a considerable bactericidal power for bacillus anthracis.

Cultures from beyond the vestibule were taken from seventy-six normal noses by Thomson and Hewlett[14] (1895) and the conclusion was reached that under normal conditions microorganisms are never plentiful. In 80 per cent of their cases the mucus was completely sterile. Klemperer[6] (1896) criticised the results of Wurtz and Lermoyez and of Thomson and Hewlett, and states that the interior of the nose always contains some germs.

Very great precautions were taken by Cobb and Nagel[24] to protect their cultures from contamination at the vestibule, and out of sixty cases five showed a few staphylococci and the rest were sterile. They were under the impression that the five cases showing bacteria were due to some as yet undiscovered fault in their technic.

Regardless of the contradictory results from the bacteriologic examination of the nasal mucus, it is a common observation of laryngologists and rhinologists that nasal and pharyngeal operations, when they do not extend deeply into the tissue, rarely present evidence of local sepsis and symptoms of systemic infection are exceedingly rare.

Analyses of the air going through the nasal passages have been made by a number of workers tending to show that very few bacteria succeed in reaching the deeper air passages. Most of them are filtered out in the vestibule about the vibrissæ.

The procedure used in collecting cultures from autopsies consisted in removing the calvarium and the brain, sterilizing the site of entrance with a glowing steel spatula, and boring an opening with a sterile trephine. Only the sphenoid, ethmoid and frontal sinuses were accessible in this manner and no effort was made to obtain cultures from the maxillary sinuses. Some of the specimens were secured by injecting sterile physiologic saline solution into the sinuses and withdrawing it. It was found, however, depending upon the position of the body and other factors, that it was difficult to recover in many cases a sufficient quantity of the solution, most of which leaked out through the ostium. The plan was adopted therefore of making the opening larger and introducing a sterile cotton swab which was tightly rolled on an applicator and passed rapidly through a flame to remove any straggling fibers. This was immediately dipped into physiologic saline to moisten the swab and smeared on horse or rabbit blood agar plates. In most cases duplicate plates were inoculated for aerobic and anaerobic cultures.

A total of thirty-three cadavers were examined, but seven have been excluded on account of the possibility of contamination or

because the sinuses were not sufficiently developed on account of age to give typical results. Of the remaining twenty-six, none were included which showed any clinical signs of sinus infection.

The causes of death relating to bacterial infection included influenzal, tubercular and staphylococcic meningitis, streptococcus viridans, septicemia, otitis media, mastoiditis and lues. There were fourteen who died of strictly nonbacterial diseases, including tumors, diabetes, bladder stones, hydropneumothorax, cirrhosis of the liver and coronary thrombosis.

The longest time elapsing between death and autopsy was 18½ hours. Eighteen of the autopsies were performed within five hours of death, five were done between five and ten hours afterward and the remainder within 18½ hours.

Table of results:

TABLE I.

AUTOPSY DATA

	Sphenoid	Ethmoid	Frontal	Total	Per cent
Number of sinuses examined	23	24	14	61	
Sinuses sterile	17	17	11	45	74
Bacteria present in	4	7	3	16	26

TABLE II.

BACTERIA FOUND

	Sphenoid	Ethmoid	Frontal	Total
Staphylococci	1	6	2	9
Diphtheroids	2		1	3
Colon bacilli	1	2		3
Streptococcus hemolyticus		1*	1*	2
Neisseria catarrhalis		1		1
Lactobacillus	1	1		2
Sarcina	1			1

*From same cadaver.

A large number of sinuses of animals were examined, including rabbits, rats, dogs and guinea pigs.

The method of collecting material from these necessarily varied considerably. In the case of the rats, the animals were killed by bleeding, the heads skinned and the fat, connective tissue and muscle overlaying the area of the maxillary sinuses removed with a pair of small scissors. By clipping off the zygomatic process the membrane lying at the root of the upper incisors was readily exposed to view. An opening was burned in it by means of a

platinum needle which was then thoroughly rubbed over the inner surface and streaked carefully on blood agar plates.

A similar method was used in making cultures from guinea pig sinuses. The method used with rabbits consisted in killing the animals by bleeding, skinning the head and removing the fat and connective tissue overlaying the triangular area below the eyes which marks the boundary of the maxillary sinus. The mucous membrane lining the sinus was readily exposed to view and an opening made by means of a glowing needle or sterile scissors. A sterile swab similar to that used in collecting material at autopsies was then introduced, rubbed over the surface and cultures made at once on blood agar plates.

An attempt was made to culture only the frontal sinuses of the dogs since it is much more difficult to enter the maxillary sinuses without contamination. They were entered by sterilizing the area with a glowing steel spatula, removing the periosteum by means of sterile scissors and forceps, and boring a hole with a steel trephine large enough to allow the entrance of a swab. These were cultured on blood agar as previously described. In every instance all precautions were taken to prevent external contamination.

Cultures were made from the maxillary sinuses of seventy rats, nineteen rabbits and three guinea pigs, and the frontal sinuses of seven dogs. All of these, so far as could be determined by gross appearance, were normal sinuses. The bacteriologic findings are given in Tables III and IV.

TABLE III.

SUMMARY OF ANIMAL DATA

	Rats	Rabbits	Dogs	Guinea Pigs	Totals	Per cent
Sinuses cultured	140	38	14	6	198	
Number sterile	126	21	10	5	162	82
Not sterile	14	17	4	1	36	18
Both sinuses sterile in animals	58	9	4	2	73	
One sinus sterile in animals	10	3	2	1	16	
Both sinuses with bacteria	2	7	1	0	10	
Per cent sterile	90%	55.3%	71.5%	83.4%		

The most frequently found microorganisms were diphtheroids and staphylococci in rats, the pasteurella group and staphylococci in rabbits, and neisseria and an unidentified gram negative bacillus

(not of the colon group) in the dogs. It will be observed that staphylococci were found quite frequently in every kind of animal examined.

Anaerobic cultures were made regularly on animal and autopsy specimens during most of the investigation but in no instance was an organism secured which was not also found in aerobic cultures. The Varney anaerobic jar was used with an incubation period of four to seven days.

TABLE IV.

BACTERIA FOUND

	Rats	Rabbits	Dogs	Guinea Pigs	Totals
Diphtheroids	10				10
Staphylococci	6	2	1	1	10
Pasteurella		14			14
Streptococci	1	1			2
Neisseria			3		3
Sarcina	1				1
Hemophilus	1				1
Gram negative bacillus (not colon)			3		3

The rabbit sinus, although normal in appearance, was found to be frequently inhabited by microorganisms of the pasteurella group. Thirteen cultures from as many different animals were taken from rabbits used for other experimental purposes in which injection of saline, alizarine, etc., were made directly into the sinuses. Only three, or 23 per cent, of these remained sterile. Although the number of examinations of rabbits is not large, the finding of bacteria more frequently in injected sinuses is in harmony with the observations of Wenner,[23] Stark[22] and others that the injection of saline, distilled water and other fluids has a pronounced effect on the ciliary activity or produces pathologic changes in the epithelial lining.

The resistance to bacterial invasion is weakened or the ability to remove promptly any foreign material is impaired.

The number of rat sinuses found to be sterile (90 per cent) is exceedingly high, as compared with rabbits (55 per cent), and much higher than in dogs (71.5 per cent). It is possible that these differences are explainable on an anatomic basis, the maxillary sinus of the rabbit affording greater probability of entrance of foreign particles and bacteria and greater probability of re-

tention after entrance has been gained. The frontal sinus of the dog, although fairly accessible to entrance, would afford better natural drainage, and more prompt removal of foreign material would be expected.

If the rabbit sinuses, in which a high incidence of bacteria occurs, be excluded from consideration, the percentage of all animal sinuses found to be sterile is raised to 88 per cent. Most of the observations were made during the winter months and some in the spring and early summer.

The writer believes the observed results, namely, the usual sterility of the sinuses, are explainable principally on a physico-anatomic basis. These conclusions are supplemented by observations on the bacteriostatic properties of sinus secretions in animals, to be reported later. They may be summarized as follows:

1. Filtering action of the vibrissæ and moist mucous surfaces of the nasal passages.

2. The combined action of cilia and gravity in washing out promptly infective particles which gain entrance.

3. The protective action of the mucous coating, which protects the mucous membrane against bacterial invasion in much the same manner as a capsule protects a bacterium from host reactions.

The vestibule and nasal passages are very efficient in their filtering action, as is shown by the work of Thomson and Hewlett,[8] and by the fact that bacteria are few in the upper passages.

The efficiency of the action of gravity and cilia in washing out the secretions and foreign particles is dependent necessarily on the anatomic configuration of the sinuses and the position of the head. The constantly shifting position of the head during life is undoubtedly an important factor in enabling the sinus to maintain itself in a sterile condition. After death the unchanging position may be such as to cause the secretions to tend to flow away from the ostium in spite of ciliary activity. Bacterial invasion under such conditions would be much easier.

The maxillary sinus of the rabbit is unusually large in relation to the size of the head. Most important of all is the position

of the sinus anterior to and below the natural opening. Foreign particles which gain entrance would therefore gravitate toward the closed end of the sinus. Only ciliary action or the maintenance of an abnormal position such as sitting on the haunches would accomplish their removal.

Gravitation and ciliary action are able to work together most effectively in clearing out the maxillary cavity of the rat on account of the location of the ostium at the lower edge of the medial wall. The small size of the cavity is a factor of importance in maintaining a normal condition, since the time required for complete removal of particles would be short and the prevention of injury to the membrane more definitely assured.

The frontal sinus of the dog occupies an intermediate position in relation to the above factors. It is large but has good drainage with some irregularities in the wall which would allow at least temporary accumulations. The ever changing position should be considered as a factor of importance in preventing the accumulation of secretions and contamination, especially in sinuses where the natural opening is above the inferior wall.

The horizontal position of man or the position "on all fours" is adapted to better natural drainage of the sinuses than the vertical position. This would indicate a failure on the part of man to adapt himself to his recent evolutionary state. The result is often disastrous, especially when some stimulus to the lining membrane causes an accumulation of fluid faster than ciliary action can remove it. The thin consistency of the fluid may render ciliary action less effective in its removal. Conditions are more favorable for bacteria and infection may result.

Experiments were performed which show that ciliary action does not stop as a result of the death of the animal directly, but only as an indirect consequence of the autolytic and putrefactive processes which ordinarily set in soon after death. Rabbit heads were prepared as usual with aseptic precautions and an opening made in one maxillary sinus through which a small piece of lining epithelium could be taken. The opening was plugged with a small piece of sterile nonabsorbent cotton and the head

placed in the icebox at 5° C. A piece of epithelium was removed for microscopic observation of ciliary activity every 24 hours.

In one experiment there was good ciliary activity after three days, the icebox being defrosted during the second night and the exact temperature not known. One membrane which was removed and placed in dextrose carbonate beef infusion broth at 5 to 8 degrees Centigrade still showed good ciliary activity after four days. Invariably all signs of activity were gone when there was the least evidence of putrefactive processes.

It was also observed that ciliary activity was completely stopped in the rabibt membrane at a temperature of 5 to 8 degrees C. and gradually increased in intensity between this temperature and 35° C. and up to 38° C., at which a maximum activity was reached. The invasion of the sinuses by bacteria soon after death, as observed by some workers, may be attributed largely to the lack of normal drainage and the beginning of autolytic processes which weaken and destroy the ciliary activity.

The statement is frequently made that the normal nasal sinus is sterile. This does not seem to be an accurate conclusion from the available evidence, since many sinuses which are normal according to usual criteria, contain some bacteria. The evidence shows that the normal sinus is contaminated by bacteria and dust particles from the air with greater or lesser frequency, depending on its anatomic configuration, but that it quickly clears itself of such foreign material.

If it were possible to examine the same normal sinuses at frequent intervals over a period of time, it is probable that bacteria would be found to be present periodically. The amount and frequency of this contamination would depend, of course, on the condition of the air which is breathed. The continued presence of large numbers of bacteria indicates a breaking down or weakening of the self-clearing activity of the sinuses.

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LXII.

SOME DIAGNOSTIC CONSIDERATIONS WHEN VERTIGO IS A SYMPTOM. CASE REPORTS.

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Greater knowledge of the anatomy and physiology of the mechanism involved in the production of vertigo will decrease the number of otoneurologic problems which continue to contribute to certain diagnostic errors. Until such time, the investigator must combine verified information with practical theoretical considerations in order that the findings of clinical observation may be practically interpreted. Such a system[5] has been projected to serve as a working unit, to interpret such disorders as arise in the intricate network of nerve pathways constituting the vestibular system.

The first part of the system, the peripheral neuron, arises in the end organ in the ampulla, includes the vestibular ganglion, and extends to the medulla, where the fibers terminate in Deiter's, Von Bechterew's and the triangular nuclei.

The second part of the system, the least understood, is comprised of the intermediate connections in the medulla and cerebellum. In the medulla the horizontal canal tract separates from the vertical canal's tract, and complex nerve connections are begun with the vestibulospinal, vestibulosympathetic, vestibulo-ocular and vestibulocerebral systems. The horizontal tract presumably divides into sensory and oculomotor pathways. The sensory pathway is thought to enter the cerebellar hemisphere of the same side through the inferior cerebellar peduncle, where it connects with the cerebellar nuclei, globosus, emboliformis and tecti. The oculomotor pathway is thought to proceed to the oculomotor nuclei, more or less directly, near the midline, through the posterior longitudinal bundle. The vertical canals tract presumably extends upward, lateral to the midline, to the level of the middle

of the pons, where it also divides into sensory and oculomotor pathways. The sensory pathway enters the cerebellar hemisphere of the same side hypothetically through the middle cerebellar peduncle to also connect with the nuclei globosus, emboliformis and tecti. The oculomotor pathway presumably proceeds more or less directly to the oculomotor nuclei hypothetically through the posterior longitudinal bundle.

The third part of the system comprises the connections with the higher centers. A nerve pathway connects the cerebellar nuclei with the sensory cortex of the cerebrum, probably near the second temporal convolution, constituting a cerebral center for sensing motion, by passing through the superior cerebellar peduncle and the crura cerebri to the cerebral cortex of both sides. Most of the fibers decussate to the opposite side.

Of the disorders of the first part of the system, it may be stated that vertigo is possible only with a functioning labyrinth, so that nystagmus to one side means that there is a pathologic labyrinth on that side, or that there is a destruction of the opposite labyrinth. Such pathology originates from hemorrhage, degeneration, neoplasms, or from serous or suppurative inflammations. The inflammatory changes are of four chief types: para-otitis, peri-otitis, internal peri-otitis with fistula, and diffuse end-otitis. Typical labyrinthine vertigo is frequently preceded by a prodrome of noises in the ear. It is combined with a sensation of turning, and there may be nausea associated with emesis. It is increased by head motion, does not stop with closure of the eyes, and there is no disturbance of consciousness. The spontaneous labyrinthine symptoms are: vertigo, nystagmus, equilibrium disturbances and past-pointing. The subjective turning is in the direction of the quick component of the nystagmus. The nystagmus is a pure reflex controlled by the vestibular system. A blind person with a turning dizziness feels an imaginary turning of the head. The spontaneous dizziness may sometimes be of a tactile character. Tactile dizziness is often found, but little is known about it—e. g., the bed seems to stand high at the foot, or the ceiling to run to the floor, and there is a feeling of drunkenness. These sensations constitute so-called "first degree symptoms of labyrinthine disturbance" before the appearance of nystagmus.

In a so-called "chronic adhesive process" with atrophy of the labyrinth, there may be adhesions in the region of the oval and round windows which either grow through both windows into the inner ear, or merely produce diminution of mobility. In either condition there is produced atrophy of both the vestibular and cochlear components. In such a process the watch test is valuable. It may be accompanied by severe attacks of vertigo. If a patient has an inner ear disease, consider arteriosclerosis and chronic adhesive process. Arteriosclerosis is usually bilateral and has an absence of labyrinthine symptoms. A chronic adhesive process is not associated with hypertension. Clinically there is continual tinnitus, slow diminution of hearing and attacks of dizziness of high degree. Labyrinthine symptoms later disappear with normal or hypoirritability, deafness is gradual, but tinnitus may not disappear unless it is well treated. After first examining the patient for choked disc to rule out tumors, lumbar puncture will sometimes be the only agency to relieve it.

Brunner[4] states that 30 to 40 per cent of climacterium patients have spasms of the vessels of the posterior fossa, giving labyrinthine dizziness or cerebellar symptoms, which are relieved by all agencies relaxing the vessels or lowering the blood pressure. It is stated that a neurasthenic patient may produce a "functional vertigo" by concentrating on memories of a previous attack. A reflex vertigo may be produced in abdominal disorders when the vestibulosympathetic system affects the blood supply of one labyrinth more than the other. Similar one-sided effects are observed in the blanching of one cerebral occipital lobe in transient hemianopia, and the blanching of one optic disc in hemicrania. Conditions in the nose and throat which affect the sphenopalatine ganglion, which has connections with the tympanic plexus, may produce vertigo by affecting unilaterally the labyrinthine circulation. Certain toxins and drugs, notably nicotin, may produce typical labyrinthine vertigo. Methyl chloride has recently been mentioned in a similar role.

When a spontaneous nystagmus of labyrinthine origin is observed, the investigator is interested in determining whether it is of peripheral or central origin. Peripheral labyrinthine nystagmus is produced by organic disease in the peripheral sense

organ. Central labyrinthine nystagmus is produced by organic disease or functional disorder in the central vestibular system. The following considerations are of importance to differentiate: both types have turning dizziness. In the peripheral labyrinthine nystagmus there is concordance in the intensity of the nystagmus and the dizziness—i. e., the more dizziness the more nystagmus, and vice versa. In central disease there may be a discordance—i. e., a marked vertigo with little nystagmus. With peripheral labyrinthine nystagmus, the patient falls in the direction of the slow component, and the direction of falling is usually dependent upon the position of the head. With central nystagmus, the patient may fall in the direction of the quick component, and the falling is, as a rule, independent of the position of the head. Acute irritative or destructive symptoms from one part of the ear alone, either cochlea or labyrinth, indicate retrolabyrinthine disease, for if either the cochlea or the labyrinth are involved in peripheral disease there are symptoms of both, while nerve or brain disorders may separate symptoms—i. e., vertigo may be present without tinnitus.

Motor labyrinthine disturbances of equilibrium vary as follows: The disturbances after functional tests are characterized by falling in the direction of the slow component, and the direction is dependent upon the position of the head. With acute suppurative diseases of the labyrinth, the falling reaction is in the direction of the slow component, and as a rule dependent upon the position of the head. With acute inflammatory diseases of the vestibular nerve and spinal acoustic root, falling is often in the direction of the quick component and as a rule independent of the position of the head. With tumors of the cerebellum and posterior fossa the falling reaction at one observation may be in the direction of the quick component and at another time in the direction of the slow component, and independent of the position of the head.

Sensory labyrinthine disturbances of equilibrium result from all destructions of the labyrinth, including deafmutism, chronic inflammation, and sequestration of the labyrinth; chronic diseases of the vestibular system, such as multiple sclerosis, syringobulbea, and chronic encephalitis, and tumors of the posterior fossa.

Vestibular characteristics of a peripheral irritative lesion of one side are: Mixed horizontal rotatory nystagmus to the side of the lesion, tendency to fall and past-point to the opposite side, vertigo, sensation of rotation toward the side of the lesion, and sympathetic responses which are pallor, sweating, nausea, vomiting and faintness, depending upon the intensity of the irritation, and, coincidental, signs of cochlear irritation, as hyperacusis and tinnitus. Neurovisceral impulses producing pallor, sweating, nausea, vomiting and faintness obviously communicate with the sympathetic system. In a depressing or blocking lesion of one side, of sudden onset, the opposite symptoms are present; or, if slow in onset, there may be produced no spontaneous symptoms. There may be coincidental cochlear depression with hypoacusis and tinnitus.

Partial loss of the vestibular reaction may be encountered; for example, stimulation may fail to elicit nystagmus, vertigo, falling and past-pointing, but the subject may manifest pallor and sweating; or another, no nystagmus and no sympathetic symptoms, but may fall and past-point. Positive reactions of any kind prove functional activity of the peripheral nerve elements of the eighth nerve and portions of the central connections of the vestibular apparatus.

Past pointing, founded by Bárány, is clinically of value only in indicating whether or not the cerebellum is diseased. No further localization is possible by this test. Pointing to the right or left is more important than horizontal variations and only clear past pointing is pathologic. It is useful in diseases of the labyrinth, vestibular nerve or cerebellum in the presence of vertigo, paresis of the arm or a combination of paresis and vertigo. For accurate past pointing the finger should come from below up to the examiner's finger. Past pointing and falling responses to stimulation are expressions of motor impulses of the vestibulospinal tract which distributes motor impulses of a general reflex character to the skeletal musculature. Past pointing corresponds to the slow component except for confusion in cerebellar abscess. If nystagmus is observed to the side of an inactive labyrinth, a cerebellar abscess may be present.

Lateropulsion, described by Babinski, is a substrata of Mènière's syndrome. It is seen in early disseminated sclerosis, and is seldom combined with spontaneous nystagmus. It gives a feeling of a force pushing to the right or left.

Vertical nystagmus is not labyrinthine unless it is combined with turning dizziness. It may be present in diseases of the midbrain (corpora quadrigemina), e. g., encephalitis or tumors; tumors of the anterior part of the cerebellum, which makes pressure on the midbrain; or abscesses and hemorrhage of the great brain, which break into the ventricle. Diagonal nystagmus is, as a rule, not labyrinthine. A retracting nystagmus has been described, in which the eyes protrude slowly and quickly retract. Exophthalmos pulsans has the quick motion out. Retracting nystagmus is observed only in disease of the aqueduct of Sylvius, such as ecchinococcus infection, or tumor of the midbrain, which grows into the aqueduct of Sylvius.

The "head motion test," described by Brunner, is less well known, and is produced by grasping the patient's head firmly, then making a quick motion backward and having the patient look quickly to the diseased side for nystagmus examination. Nystagmus is observed by this method in paralabyrinthitis, disseminated sclerosis, skull injuries (traumatic neurosis), some cerebellar tumors and ecchinococcus infection of the fourth ventricle. In disseminated sclerosis, nystagmus can be easily produced by this method if not already present. The differential point of the test is this: in paralabyrinthitis the test cannot be repeated, the others, being central diseases, have a nystagmus which lasts a long time and the test can be repeated. With the disorder in the labyrinth one may differentiate as follows: With a cholesteatoma making pressure on a window, particularly the oval window, the head motion test is negative; in bone affection without fistula, i. e., labyrinthitis, the fistula test is negative and the head motion test positive; with fistula the fistula test is also positive. With this test one can diagnose a paralabyrinthitis.

In lues it is frequently found that the horizontal canals alone are destroyed, both sagittal canals reacting. The fistula symptom without a fistula is a distinct symptom of hereditary lues of the

otosclerotic type with a red promontory. It may be produced by a relaxed annular ligament or by an encephalitis of the brain stem produced by the hereditary lues.

Hyperirritability of the vestibular system is found in paralabyrinthitis, neuritis of the vestibular nerve, cerebellar tumors, traumatic conditions following skull injuries and syringobulbea. Serous labyrinthitis may very early produce hypoirritability, but paralabyrinthitis always produces hyperirritability. Destruction or irritation of the vestibular or cochlear nerve alone localizes the disease behind the labyrinth. Cerebellar tumors give the highest degree of caloric nystagmus, sometimes sustained for from four to five minutes, associated with slight vertigo. In traumatic conditions following skull injuries vertigo is the usual symptom. The head motion test will bring out the nystagmus. Syringobulbea gives normal or hyperirritable symptoms. An interesting combination of nystagmus with recurrent laryngeal paralysis on the side of the nystagmus may be observed in a patient who has been treated for gastric disturbance because of the central vagus involvement. This combination is observed in acute disorders of the arteria cerebellaris inferior posterior, particularly with emboli or hemorrhage, encephalitis of the medulla, or in syringobulbea which is slow in onset and has a nystagmus most marked on the diseased side.

Hypoirritability of the vestibular system is present in diseases of the nerve such as neuritis or trauma with the hearing destroyed and the caloric and turning tests negative, and neoplasms with complete destruction of the posterior longitudinal bundle, and with so-called "reflex nonirritability." Of the neoplasms, angle tumors are most important, giving complete deafness, choked disc, complete nonirritability, absence of corneal reflex, spastic symptoms and at times abducens and facial palsy. The choked disc is the important symptom, for the other symptoms might be produced by disseminated sclerosis. Hemicrania may produce these symptoms with some edema of the papilla, but only unilaterally, and the symptoms disappear after the attack. Absence of corneal reflexes is one of the most common symptoms but may often be found only by Oppenheimer's method of demon-

strating it—i. e., if the patient lies on the diseased side the corneal reflex disappears on the diseased side for tumors of the cerebellopontine angle, if mobile, press on the fifth nerve and abolish the reflex in this position, but not if the patient is recumbent. There is seldom facial palsy in angle tumors, for the nerve is quite resistent. Tumors of the pons may arise, starting with sensibility disturbances, later giving angle tumor symptoms. Angle tumors, if not operated upon, lead to deafness, blindness and dizziness.

Complete nonirritability results from complete destruction of the nerve, the longitudinal bundle or the whole spinal acoustic root. No disease but multiple sclerosis produces complete destruction of the spinal acoustic root. Longitudinal bundle destruction may occur with tubercles, encephalitis or gummas of pons, for these fibers are compact and easily damaged. Reflex nonirritability results from tuberculosis or latent cholesteatomatous labyrinthitis or from angle tumors. In Von Recklinghausen's disease it is not uncommon to have tumors in the eighth nerves, which are soft fibromas. The differential points from angle tumors are that the patients are mentally subnormal, there are tumors in other places, and the hearing is always preserved.

Dissociation in labyrinth tests offers some diagnostic information. In the normal individual, response is given to the galvanic, caloric and rotation methods. Abnormalities of interest are these: The galvanic response only is present in all suppurative diseases of the labyrinth, hereditary lues and traumatic destruction of the labyrinth. The turning and galvanic responses only are present in serous labyrinthitis, hereditary lues, head injuries and toxins, especially carbon monoxide. The caloric and galvanic reactions only are present in acquired lues.

Dissociations in labyrinthine reactions are of interest. Only nystagmus may be present in normal children, rarely in adults with nonlabyrinthine nystagmus, lues, traumatic neuroses and occasionally in cerebellar tumors. Nystagmus may be absent, the falling and past pointing reactions present, and dizziness present in neuritis—e. g., nicotine poisoning and cases where the longitudinal bundle is destroyed.

If a patient exhibits an ocular nonlabyrinthine type of nystagmus, after rotation he will walk in the direction of the slow component which he should have. Only special cases require the turning chair. The caloric tests are usuallly sufficient and produce nystagmus but little vertigo. In the rotation tests, the horizontal tests usually suffice, are less painful and take less time. In caloric testing, some diseases react to 5 cc. but not to larger quantities—e. g., hereditary lues and healed radical operations. The use of caloric stimulation has one great advantage over the use of rotation for some tests, in that it permits stimulation of one side only. In douching the right ear, for instance, with the head 120° forward or 60° backward, only the right horizontal canal is stimulated, or with the head 30° forward only the right vertical canals, according to the choice of the investigator, a selective stimulation of the canals of one side obviously impossible when rotation is used as a stimulus.

Coordination disturbances are distinctly cerebellar and not labyrinthine. The rebound phenomenon of Holmes, Stewart and Sherrington is demonstrated by having the patient's elbow flexed, attempting to push the examiner away. When the examiner suddenly lets go, the arm shoots froward and normally rebounds but not in a patient with cerebellar disease. The side having the abnormal rebound phenomenon is the side of the lesion. Orientation of the head is regulated and coordinated by the cerebellum, which cannot produce nystagmus but regulates its intensity. The symptoms of labyrinthine suppuration that warrant suspicion that the cerebellum is involved are spontaneous nystagmus with vestibular dizziness and falling associated with either headache or fever. These symptoms indicate an involvement of the posterior fossa by meningitis, or abscess or both. The vestibular manifestations are frequently present in cerebellar abscess. The nystagmus, vertigo and falling of acute labyrinthine suppuration resemble somewhat the symptoms resulting from pressure on the brain stem. In cerebellar abscess it may be impossible to distinguish between them, particularly since both processes are in most instances coexistent.

In demonstrated lesions involving the cerebellar hemispheres and in involvements at or near the second temporal convolution

in the cerebrum the usual sensory signs of stimulation of the end organ are absent. This suggests a tract from the cerebellar nuclei for muscle sensing. In demonstrated lesions at or near the midline at the level of the pons there is an absence of rotatory nystagmus on stimulation of the vertical canals. This suggests that a tract connects the nuclei of the medulla to the oculomotor nuclei.

Dissociation of ocular movements is of diagnostic value. Conjugation is affected by the coordinating oculomotor brain, consisting of the posterior geniculate body, the corpora quadrigemina, the third, fourth and sixth nuclei and associated nerve connections. Under certain abnormal conditions conjugate movements may be present on normal gaze, but with stimulation of the end organ nystagmus is produced characterized by dissimilar movements of the eyes—e. g., one eye may show horizontal nystagmus while the other shows vertical nystagmus. This suggests a lesion in the so-called "oculomotor brain." Normal nystagmus movements produced by stimulation of either side are similar in direction, rotation, plane and extent in the two eyes, which proves that the motor tract from each vestibular end organ is more or less directly concerned with oculomotor coordinations. Dissociation of ocular movements due to disturbed oculomotor coordinations is not due to lesion along the motor tract but to lesion in the oculomotor centers, because oculomotor incoordinations are not developed in demonstrated lesions at various points along the vestibuloocular tract, but there is a development of inverted, perverted or incoordinate nystagmus in demonstrated lesions at or near the midline in the region of the oculomotor centers.

When the lesion has a supratentorial location the following vestibular characteristics are found: Past pointing and falling are less marked than normal; vertigo is characteristically slight or absent, either spontaneous or from stimulation; nystagmus is of slow rate and wide amplitude after stimulation; and spontaneous ocular movements tend to conjugate deviation.

When the lesion has an infratentorial location the following vestibular characteristics are found: Spontaneous pallor, sweat-

ing, nausea, vomiting and faintness are frequently associated with infratentorial or peripheral lesions, but in the infratentorial lesions it is impossible to increase these symptoms by moderately intense stimulation; falling and past pointing reactions are exaggerated; spontaneous vertigo, especially if sharply defined as to plane and direction, is exaggerated in infratentorial or peripheral lesions; and perversion of nystagmus may be produced instead of normal nystagmus. The spontaneous nystagmus is rapid and of small amplitude, and, unlike that produced by peripheral lesions, is not mixed horizontal rotatory, but horizontal, vertical or dissociated.

Case No. 1 (An irritative lesion).—Mr. B. T. B. was admitted to the Evanston Hospital January 25, 1929. The complaint upon admission was dizziness. One month previous to admission the patient suddenly developed severe pain in the right side of the head, principally back of the right ear. No ear infection was present. Following the initial attacks of pain there were several attacks of vertigo. There was at no time a loss of consciousness. He was able to reach a chair or bed with little difficulty. The attacks were characterized chiefly by blurred vision and extreme vertigo. There was no tendency to fall to a particular side, there was no nausea or vomiting, and no recurrence of the headache. Four days before admission he had a very severe attack of vertigo while at his desk; he felt quite weak and his associates stated that he was very pale. There was a daily recurrence of these attacks until he was admitted to the hospital. He stated that he had enjoyed general good health until the present attacks. The family history was essentially negative. A neurologic examination was made by Dr. Bassoe, the first day after admission. The pupils were equal and regular. The discs were normal. The deep and superficial reflexes were normal. Wassermann and Bárány tests were advised, with complete rest, and no tobacco. The Wassermann test was negative. The writer observed that the external canals were unobstructed and the drum membranes were intact and normal. The vestibular tests were given the second day after admission. These tests were done in the hospital by caloric stimulation with the patient at rest in bed. Water at 10° C. was used. Irrigation of the right ear with

30 cc. gave a horizontal rotatory nystagmus to the left of normal amplitude, after 45 seconds, lasting 60 seconds. Irrigation of the left ear with 30 cc. gave a horizontal rotatory nystagmus to the right of increased amplitude, after 30 seconds, lasting 90 seconds. This was interpreted to mean a noticeable increase in the irritability of the right labyrinth. The patient continued to improve in his freedom from vertigo and was sent home after five days to plan an extended rest in Florida. Two days after being sent home he was seized with a violent vertigo at night and died ten minutes after readmission to the hospital.

Necropsy report (Dr. Jenkins): "There is a clot distending the right vertebral and basilar arteries. It is molded to fit the lumen. It does not cling to the wall. It is dark purple and soft. The wall of the basilar artery is slightly stiff. Its lining contains firm yellow elevations. The first portions of the anterior and middle cerebral arteries contain only fluid blood.

"Microscopic examination of the vertebral and basilar arteries: The wall of the basilar artery is greatly thickened, most of the thickening apparently being in the subintimal region, where there are many angular spaces which have probably contained cholesterol. There are very large regions of calcification. There is an annular thrombus almost completely occluding the lumen, only a small channel remaining in which there is fresh or recently clotted blood. The thrombus is in varying stages of organization, and some of it is infiltrated heavily with lymphocytes and leucocytes. The wall of the vertebral artery is not markedly thickened, but its lumen is almost completely occluded by a thrombus similar to that in the basilar artery but more completely organized. The blood clot from the anterior part of the basilar artery consists of recently clotted blood in its center and of fibrin and well preserved leucocytes at the periphery.

"Complete anatomic diagnosis: Recent and organized thrombosis of the basilar and right vertebral arteries; sclerosis of the lining of the coronary, vertebral and basilar arteries; nodular encapsulated, fibrous and caseous tuberculosis of the left kidney; left fibrous pleuritis."

Case No. 2 (a blocking lesion).—P. K. was admitted to the Evanston Hospital July 9, 1929. This patient had recovered from a bilateral otitis media associated with a scarlet fever, only to be precipitated into another bilateral otitis media by an attack of measles. Upon admission to the hospital, two weeks after the onset of the second attack, the temperature was 105°, the leucocyte count was 16,500, of which 78 per cent were polymorphonuclear cells, there were typical symptoms of a right acute suppurative mastoiditis, a marked right cervical adenitis and a subsiding left otorrhea. Roentgenograms revealed suppurative changes in the right mastoid but no changes in the left. The right mastoid was opened on the day of admission, the lateral sinus exposed and found unchanged, and the dura not exposed. On the fourth postoperative day the cervical adenitis appeared to be undergoing suppuration and, under gas anesthesia, an incision was made which yielded about 20 cc. of yellow pus. On the eighth day there was a distinctly phlegmonous state in the right cervical region and, under anesthesia, three deep incisions were made but no pus found. The cervical reaction decidedly retarded healing of the mastoid wound. On the twelfth day pus came from the deep incisions, and by the fourteenth day there was a striking local improvement, but the patient looked extremely toxic. The nurse stated that the patient had complained of hearing foreign sounds. The hearing was noticeably diminished. There was a slight horizontal rotatory nystagmus on looking to the extreme left, the left drum membrane was intact with no bulging and there were no symptoms referred to the left ear. The knee jerks were brisk, there was a bilateral ankle clonus, slightly more pronounced on the left at times, a questionable bilateral Babinski and no Kernig. On the eighteenth day, with all local symptoms clearing rapidly, the temperature, pulse and respiration dropped to normal but the patient was moribund. The spinal fluid revealed 800 cells per cubic millimeter, but was not under noticeably increased pressure. The white blood count revealed 35,000 cells per cubic millimeter, of which 76 per cent were polymorphonuclear leukocytes. In the absence of further localizing symptoms and because of the serious state of the patient, no exploratory

work was done. The patient expired on the twentieth postoperative day.

Necropsy findings revealed no right sided intracranial pathology. There was no meningitis. Opening of the left temporal bone revealed an intact drum membrane, softening of a few cells high in the petrous portion with an erosion at the internal auditory meatus. There was an abscess stalk from this point of contact to the adjacent cerebellum and nearly the entire left cerebellar hemisphere was filled with pus.

COMMENT.

The patient with the thrombosed right vertebral and basilar arteries had a transient extreme vertigo with an experimental nystagmus indicating only a slightly increased irritability of the right side. This discordance and the absence of acute ear pathology point to a central lesion. The unsteadiness and tendency to fall during the attacks, without any tendency to definitely fall to the right or left, are suggestive of a cerebellar involvement. The horizontal rotatory nystagmus and the conjugate eye movements with equal pupils preclude any lesion of the oculomotor brain. An infratentorial location of the lesion was indicated by the extreme vertigo and the spontaneous pallor, sweating and faintness. A nicotine poisoning can very nearly simulate these symptoms.

The patient with the cerebellar abscess had a slight tinnitus, increasing deafness and slight nystagmus on looking to the extreme left, which would undoubtedly have merited more attention had not the presence of so much pathology on the opposite side and the contraindications to vestibular testing masked the situation. The left vestibular and cochlear nerves were blocked by the adjacent necrosis, but the left cerebellar abscess would account for a continued slight left nystagmus.

CONCLUSIONS.

In every lesion of the brain, particularly in the posterior fossa, vestibular tests should be utilized as an aid to diagnosis in the absence of contraindications, but the diagnosis should not be

made on these tests alone. Many interesting otoneurologic problems may reach solution by applying knowledge concerning the vestibular apparatus and by otologic and neurologic collaboration.

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636 CHURCH STREET.

LXIII.

A STUDY OF THE COMPLICATIONS THAT MAY FOLLOW MYRINGOTOMY.

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In theory, myringotomy, to the otologist, is simplicity itself. It is merely cutting through a thin membrane into a pus pocket. It is so simple that almost any physician or surgeon feels competent to perform this operation with the same abandon that the oculist lances a hordeolum. It is so simple that we teach our junior medical students the landmarks of the tympanic membrane and the technic of incising it in acute otitis media.

In practice, myringotomy is not always so simple. The majority of otitis media cases are in children who bear pain poorly; the external auditory meatus demands bright illumination in order to make the tympanic membrane visible, and thus assure precision of technic in incising it; the anatomic landmarks, so easily described in a normal ear, may be entirely obliterated; all these at times make what seems a simple operation one of real difficulty. Added to the above are the facts that, in the middle ear there are nerves, blood vessels, and the delicate organs that play a part in conducting sound waves, any one of which may be injured by faulty technic, which make the operation one that is still more complicated. Furthermore, from the middle ear there are tracts that lead to the brain, and on the inner wall is a delicate membrane which guards the portals through which infection may be carried. A knife, therefore, in the hands of the unskilled operator, may incise this membrane and open the pathway so that infection may be carried by lymphatics and blood vessels to the delicate organs within the skull. Thus the idea, often taught, that myringotomy is always simple, may have to be revised.

Personal experience and the observations of others suggest the possibility of doing damage in a myringotomy. One patient on whom the operation was done under unfavorable circum-

stances, became dizzy immediately afterwards, and was thus troubled for some months. On another patient, a case of acute otitis media, myringotomy was performed, which terminated in a brain abscess. In the first case, it appeared to me there might have been a dislocation of the stapes; in the second case, I was led to believe that an incision of the membrane on the inner wall of the middle ear may have been the opening through which infection gained entrance to the brain. In a third case, myringotomy was performed and was followed by profuse hemorrhage, so marked as to suggest that the operator had opened the jugular bulb or the carotid canal. Such cases merely indicate the possibility of complications, especially if there are errors in technic, or in certain cases where there are anatomic abnormalities present. When one considers that the inner wall of the tympanic cavity is not more than one-half inch square, and that there are a half dozen or more pathways through it from the middle ear to the brain, it is better understood why great care should be taken not to open any of these avenues.

Our purpose now is to review the areas which may be injured by a myringotomy, and the tracts through which infection may pass from the middle ear to the brain.

Let us now tabulate some injuries that may be produced in doing a myringotomy which may lead to unfortunate results:

1. Severing the chorda tympani nerve may pass unnoticed by the patient, and yet he may observe that he cannot taste on the side of the tongue on which the myringotomy was performed.

2. The round window is usually so far back and so well protected that it is not injured, but in some cases it may be punctured by making a deep incision far back. What the end results may be when an instrument is passed through a pus pocket and through the round window into the perilymph of the labyrinth needs no comment.

3. Severing the stapedius muscle is also possible, even though that muscle is very short. Because of the close proximity of the severed ends, they may reunite, but even then there is probability of the hearing being affected.

4. Due to the dislocation of the stapes in the oval window, or tearing it away from its articulation, a possible injury may be

accompanied by symptoms similar to those occurring after damaging the round window.

5. The jugular bulb is normally located posterior and inferior to the tympanic cavity. Injury to this may cause a hemorrhage that is embarrassing.

6. The carotid canal is anterior to this cavity, and injury here may prove very serious. Two patients have come to my attention; and in each instance myringotomy was followed by a hemorrhage; one of them fatal. Either the jugular bulb or the carotid artery must have been injured.

7. The epithelium in the tympanic cavity is in part columnar and ciliated with small cells between the ciliated cells.

When the epithelium covering the mucous membrane on the inner wall of the tympanic cavity is damaged by an incision from a knife which has passed through a pus pocket, an opening is made for the passage of an infection to the brain. As long as the epithelium is intact, it is almost impossible for infection to get within the skull. The epithelial cells are the shock troops that guard the entrance to passageways to the brain.

With these possibilities of injury in mind, let us now review the tracts through which infection may be carried through the skull to the brain after the barriers that guard the gateways have been broken down. Through injuries to the round window and oval window, infection can be carried to the perilymph into the bony labyrinth. Also, between the round window and oval window, and below the pyramid from which the stapes muscle arises, is a small depression, the sinus tympanicus, which indicates the position of the ampullated end of the posterior semicircular canal and is perforated by one or two minute foramina for blood vessels. Infection may be carried into the bony labyrinth, and from the labyrinth into the intracranial cavity through the following channels:

A. "The aqueductus cochlea, a canal measuring 10 to 12 mm. in length, and opening on the under surface of the petrous part of the temporal bone in front of the fossa jugularis. This aqueduct establishes a communication between the scala tympani and the subarachnoid cavity and transmits a small vein to join the inferior petrosal sinus."

B. A narrow canal, "about 8 mm. long, called the aqueduct to the vestibule, passes backward from the vestibule and opens on the posterior surface of the petrous portion of the temporal bone into a small slit located about half an inch lateral to the opening of the internal auditory meatus. The slit is overhung by a thin layer of bone. The canal transmits a tiny artery and vein, and a delicate membranous tube, called a ductus endolymphaticus, which emerges from the external aperture of the canal and dilates into the form of a small bag, called the saccus endolymphaticus, which lies under cover of the dura mater."

3. The internal auditory meatus is about 10 mm. long, and from the internal opening extends laterally and downward into the bone, and ends blindly, but is perforated at the end. The nerves which supply the semicircular canals, vestibule and cochlea, pass through some of these perforations. Infection in the bony labyrinth can thus get into the internal auditory canal, and through it into the cranial cavity.

4. The subarcuata fossa is a small ill-defined depression about midway between the opening of the meatus and the aperture of the acqueduct, but at a higher level than either of them. It is larger and better defined in the skull of a child than of an adult. Small vessels pass through its floor into the bone.

The mucous membrane covering the inner wall of the middle ear protects all these avenues from infection. A wound inflicted in this membrane may be the source by which infection may be carried through certain of these passageways into the brain. If by a myringotomy, infection is carried into the jugular bulb, a resulting thrombosis may carry infection into the brain. If the infection is carried from the middle ear into the carotid canal, another large passageway is entered through which bacteria may gain entrance to the meninges.

In the case of otitis media the physician usually sees the patient several hours after, and in some instances several days, after the first symptoms of otitis media have been manifest, and after solicitous friends have used various oils, salves or other remedies in the external auditory meatus. Often cases present themselves in which so much débris is found in the canal that the tympanic membrane cannot be seen. This may be removed by irrigating

the canal with warm soda solution, care being taken not to use force enough to increase the pain. After the canal is cleansed and the tympanic membrane made visible, one may find all anatomic landmarks obliterated. Because of this, it is all the more important that the physician have a familiarity with the topography of the tympanic membrane and its relationship to the external canal. One may attempt a myringotomy and be embarrassed later to find that the posterior canal wall has been punctured near its junction with the tympanic membrane.

Before doing a myringotomy the field must be sterilized as thoroughly as possible, because of the danger of carrying other bacteria from the external canal into the middle ear and thus creating a mixed infection. Many methods are used to sterilize the field. Personally, I dislike anything that discolors, such as mercurochrome or iodin, for the discoloration causes confusion when one looks at the inflamed and bulging membrane. Alcohol is a more satisfactory agent.

Much care should be taken in choosing the anesthetic and instruments in performing myringotomy. Both local and general anesthetics have their advocates. Children should always be put to sleep, either with nitrous oxid or ethyl chlorid, as this assures an absolutely quiet patient and one free from pain when the work is done. This gives the surgeon a chance to operate with such caution and deliberation that he can do the work with less danger of injuring any part of the middle ear. It also relieves the patient of a tense mental strain, and at the same time obviates acute pain. In adults, one may use local anesthetic, but even here it is better to use gas.

The source of illumination is of paramount importance. A head mirror may be used with various methods of light, such as kerosene lamps, electric bulbs, Bunsen burners, etc., also electric lighted headlights. However, I feel that the best light is from an electric lighted otoscope. The advantages in using this instrument are:

1. The source of light is such that it is easier to focus on the field of operation.

2. There is a magnifying glass which more clearly reveals the operative field.

3. In using a head mirror, whatever the source of light, the head of the patient has to be moved in order to view all regions of the field. With an electrically lighted otoscope a slight movement of the instrument exposes all surfaces.

The operative field should be lighted as much as possible. In eye work the field is flooded with light in order that the surgeon may constantly keep his knife under observation and not injure the lens of the eye. Here the surgeon has the use of both eyes, which better enables him to measure distances and depths. In the ear, he is using only one eye, hence the greater need for good illumination. The light must be good, and it must be focused on the operative field.

Care should be shown in selecting a knife. This instrument should be thin and as pointed and sharp as a cataract knife. No more pressure should be used in penetrating the tympanic membrane than is used in cutting through the cornea. Where a dull pointed knife is used, the blade may suddenly penetrate the membrane, pass through the pocket of pus and injure the lining membrane of the inner tympanic wall.

Where to make the incision is a matter of no small consideration. Some make a puncture in the part most bulging. Some begin at the lower part of the posterior inferior quadrant and make the incision upward, while others begin at the upper part of the posterior quadrant and cut downward. I prefer the latter method because the angle of the tympanic membrane is such that after pushing the knife through and drawing downward it will naturally emerge without danger of injuring the inner wall or any of the danger points above mentioned.

CONCLUSIONS.

1. A myringotomy may cause serious complications.
2. One should be familiar with the anatomic landmarks of the tympanic membrane.
3. One should know the sites which might be the source of complication.
4. The very best illumination of the operative field should be secured.
5. A sharp pointed knife with a sharp blade should be used.

6. It is better that the patient be sound asleep.

7. As much care should be used by the aurist not to strike the inner wall of the middle ear as is used by the oculist not to injure the crystalline lens when working on the eye.

Finally, the suggestion is made that in performing autopsies, on cases of acute otitis media in which a myringotomy has been performed, the mucous membrane on the inner wall of the middle ear should be examined for evidence of trauma, and each portal through which infection might be carried to the brain should be investigated.

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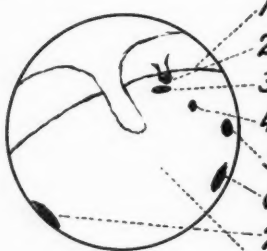
DANGER POINTS IN DOING MYRINGOTOMY



1. Chorda Tympani Nerve
2. Stapedius Muscle
3. Oval Window
4. Sinus Tympanicus
5. Round Window
6. Jugular Bulb
7. Carotid Canal
8. Mucous Membrane Covering Internal Wall of Middle Ear

LXIV.

IMPORTANT POINTS IN TONSILLECTOMY.*

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Careful observers have long since decided strongly in favor of tonsillectomy versus tonsillotomy. Sluder says at present there is almost no one advocating tonsillotomy. Yet the 1930 Year Book contains the startling statement that 73 per cent of the tonsil operations are incomplete, and it is also authority for the statement that more germs per kilogram are found in the remnants than in unoperated tonsils, hence they are far more dangerous than they were before the mechanism of drainage had been interfered with. Speaking of percentages, it would be of immense interest to know what percentage of these vicious tags occur in operations performed by laryngologists. It would be shocking as well as startling because more is expected of us, and our work is inspected more critically. Many of our cases are referred by careful internists, pediatricians, etc., seeking the complete removal of focal infections. Is it fair to them to return their patients in a far more dangerous condition than when we received them? An editorial note in the Year Book says, "Time has come when a wave of conservatism regarding tonsil operations is due. The tendency of general surgeons, pediatricians and general practitioners to undertake operations in a special field like laryngology should be deprecated." We can best do this by improving our work and thus raising the standard. In studies of postoperated tonsil patients for immediate and remote results, complications, etc., the presence or absence of stubs should be noted, though it is the exception that this is mentioned. It would appear that often failure to achieve desired results is due to the incompleteness of the operation. Is there anything that can more completely bring

*Read before the Colorado Congress of Ophthalmology and Oto-Laryngology, August 1, 1930.

the tonsil operation into disrepute than to leave so large a percentage of our patients not only unimproved by operations, but through our own efforts render their focal infections more harmful to them?

In my work at the Research Diagnostic Clinic, I often feel greatly chagrined to have the patient state emphatically that his tonsils are out because they were removed by a specialist, only to find a miserable looking throat, with badly infected stubs and considerable scarring. These patients come from a wide area; the condition exists all over the country. We have the privilege to demand tonsil work only as we merit it, by at least reversing our figures to 73 per cent of perfect postoperative results instead of the 28 per cent or less as it stands today.

This is not a plea for the adoption of any definite method of tonsillectomy, although I wish to make a few suggestions along the line of procedure which will make for a complete removal of the tonsil and its surrounding tissue element, called its capsule, leaving a fossa completely lined with fascia, the muscular bed intact. A more thorough understanding not only of the gland itself but its surroundings will aid us in developing our technic.

There are two elements to consider in our dissection. One, the capsule which covers the attached part of the gland; the other, the pharyngeal fascia, covering the entire mass.

The tonsil is suspended in its muscular bed by the folds of this pharyngeal fascia, which I have called the suspensory ligament, in an article read in 1920 before the American Academy of Ophthalmology and Otolaryngology. The relationship of gland and fascia is not competely described in the literature, although it has been mentioned, in whole or in part, by different men, from whom I shall quote freely later. In this article the tonsil will be spoken of as the entire lymphoid mass with its investment tissue entirely attached.

The capsule or attached surface is modified for a special function, perhaps two of them, one of which may be a mechanical filter, the other its power of reproduction. This power of reproduction has been assigned to the capsular tissue as well as to the

lymphoid tissue. Doubtless both have it, and for that reason I advocate the complete removal of all capsular and lymphoid tissue. I wish to make some quotations to show how the capsule has been treated with respect by some, and by others denied an existence after they had given it quite honorable mention. Makuen says, "The tissue called the capsule is the lower part of the second pouch from the third branchial arch and in some unknown way possesses the power to produce and reproduce itself, as well as lymphoid tissue." He also says that he is convinced that the capsule does not exist as a definite entity and that some of us go so far as to deny its very existence, terming it an artefact. And in speaking of the intra- and extra-capsular tonsillectomy, says, "The intracapsular leaves most of the capsule to line the fossa." Patterson of Cardiff, England, says in speaking of the capsule, "Like the larvæ which spins its own cocoon, the tonsil may be said to weave or add to its own capsule." Barnes, in his volume, "The Tonsil," speaks of the loose layers of the capsule, and throughout his book the capsule is mentioned many times. Poynter considers the capsule as a condensation of the loose mesenchymal tissue that surrounds the tonsil and separates it from the surrounding musculature. Then he says that he is not able to demonstrate it as a definite entity. Fowler, in his most excellent work, "Tonsil Surgery," says, "The capsule covers about four-fifths of the entire gland." Then he joins the ranks of the phantom chasers, saying the surgeon creates it as an artefact by stripping away layers of fascia. I wish to stress not only the existence of the capsule but its importance, and to state most emphatically that if we overlook even a small bit of it, we will later have to admit it as a definite entity, to our chagrin. If the dissection is dry we can easily detect these tags of capsule or lymphoid tissue remaining in the fascia lined fossa, and in removing stubs the second, third or even fourth time they will be found completely surrounded in their new bed by this same limiting membrane and in such a shape that it was impossible to have been created by the skill of any artisan into so complete an artefact at either operation.

The pharyngeal fascia is the second element for consideration. Dr. Hall says: "Let us consider the tonsillar mass as a herniated

lymph node and this fascia being the cul de sac surrounding it. It is folded over much as a sac might be closed over a ball by drawing the sides together and twisting them slightly. Through the folds of this fascia are found all of the blood vessels, lymph channels and nerves that actually enter the tonsillar mass."

Again quoting from Dr. Patterson: "The intrapharyngeal fascia extends from the base of the skull to the region of the esophagus surrounding the tonsillar mass, or as the tonsil developing in the recesses of this fascia." This is the nearest approach we find in literature to a description of this fascia. Let us trace the direct relationship of this membrane to the capsule as well as to the entire gland. The first appearance of this fascia is seen in the plica triangularis, which Fetterolf says, "springs from the free margin of the anterior pillar." Barnes says: "For the purpose of the surgeon it should be borne in mind that in the mature tonsil the plica triangularis is part of the capsule and not a part of the anterior pillar, as I once thought." He says: "The plica triangularis arises from the free posterior margin of the anterior pillar. Its apex blends with the soft palate above." Hammar says: "The plica triangularis is an early development of the tonsil and marks the division between the anterior and posterior outgrowths; somewhat later it contains a greater or less amount of lymphoid tissue." Thus he considers it a part of the tonsil. Poynter says: "The plica triangularis is one of the three principal foci for the development of the lymphoid tissue, and they are all within the capsule." So much for the plica triangularis. Now there is another fold of this fascia, a fold meeting the plica triangularis more or less at right angles, on the posterior surface of the tonsil. It has been called the plica transversa. It is spoken of by Gruenwald as the division of the two masses, upper and lower lobes of the tonsil, and he says it may remain more or less well marked as a fibrous fold. And in speaking of this fold of the fascia, Sluder says: "Keeping in one's mind's eye the central mass or masses growing out of the tonsillar sac, one early follows the formation of the retrotonsillar plica which is a fold of mucous membrane attached to the posterior pillar and extends more or less over the tonsil." Hett has noted that in the usual method of

enucleation, the lower region of the tonsil is in very close relation to the muscle, due to a narrowing of the areolar space, and that one is very apt to remove some of the muscle. This, by the way, is the crux of Dr. Fowler's "Tonsil Surgery." He terms it the muscle free dissection of the tonsil and says that is our object in a complete removal of the gland. One cannot place too much emphasis on this point. His great work will do much in elevating tonsil surgery.

The suspensory ligament, or the three point suspension of the tonsil, are the main points around which the operative procedure hinges. The object is to trace this pharyngeal fascia throughout its foldings and infoldings as related not only to the capsule but to the entire tonsillar mass. Its first appearance is as the plica triangularis, which in its passage over the crown of the tonsil comes to a point turning upon itself at the outer superior angle of the gland, follows the line of mucous membrane attachment, paralleling it at a distance of about one-quarter of an inch posteriorly. It proceeds toward the posterior pillar a variable distance, according to the size and shape of the gland; turning backward and downward, it bisects the posterior capsular surface of the gland. It spreads out as a fan shaped fascia, much as the mesentery surrounds the gut, its outer edges mingling with the muscle bed. This marking often remains in a throat after operation, and at times it is so prominent as to give the appearance of three pillars when the tongue is depressed toward the median line. Below, the capsular border mingles with the plica transversa. The plica transversa's anterior point of attachment, though at times very thin, can always be determined and its fibrous separation from the true capsule noted. Here sharp dissection is quite essential, especially in starting. Sense of touch has no place in tonsil dissection where the sense of vision can be relied upon. The points can best be considered by describing the operation, the technic of which is briefly as follows:

The anesthesia plays an important part in the operative procedure performing at least a partial hydraulic dissection. Since giving up the use of cocain the technic of anesthesia has been somewhat changed and is briefly as follows: The blunt needle

is still used. The first injection is made at about the junction of the posterior pillar and the supertonsillar arch just above the line of attachment to the mucous membrane. The pressure of the needle lifts this point upward and backward until it penetrates the covering. As soon as the eyelet is buried from view 1 cc. of 1 per cent novocain solution with suprarenin is rapidly introduced, the eyelet toward the tonsil. The bluntness of the needle pushes the capsule ahead, as it is sharp enough to dissect the capsule but not through it without considerable pressure. The fluid fills the peritonsillar space surrounding the posterior bilateral surface of the tonsil; in other words, the perpendicular half in contact with the posterior pillar. The second injection is made just above the attachment of the posterior border of the anterior pillar of the tongue. The tongue is depressed just enough to show this area; then it is held slightly toward the median line. As a rule, if the plica triangularis is not too prominent, a triangular sulcus, base downward, is shown between the tonsil and anterior pillar. The needle is inserted at the inner side of this triangle against the tonsil. The point coming in contact with the capsule dimples it, as before described. As soon as the needle penetrates the thin mucous covering, it seemingly falls into a cavity under the upper lobe. The injection is started and the point of the needle is made to burrow under the tonsil as though it were prying upward and forward. Here again 1 cc. of 1 per cent novocain solution is expelled rather rapidly against the capsule as the needle progresses. The solution enters the peritonsillar space here better than at any other point, as this seems to be the parting of the ways of the muscle and the gland. There is no indication at this time that the solution has entered the tissues, as there is no blebbing or elevation of the tissue and only a blanching which occurs after a few moments. The fluid is absorbed from the peritonsillar space into the tissues, not forced into them. This is not an infiltration anesthesia. About ten minutes is required for complete anesthesia, as novocain acts more slowly by topical application than cocain. The object of the injections thus placed is to have them enter the space behind the tonsil, one on each side of the ligament which bisects perpendicularly this area. Peritonsillar abscesses are subjected to this same walling off. In

the third injection the needle is inserted into the center of the mass of lymphoid tissue below the tonsil. From one-half to 1 cc. of the solution is used, depending upon the size of the mass. This injection can be termed an infiltration anesthesia. If there is but slight amount of this lymphoid tissue, or if for any reason it is not to be removed, the third injection may be omitted.

The tonsil is held upward and toward the anterior pillar with the Boettcher hook or the dissector, whichever serves to give a more direct exposure of the line of attachment of tonsil to posterior pillar. Certain types of tonsils can be displaced, showing this line of attachment as the bottom of a groove. A Bard-Parker knife, with the point of the blade having been removed and the end sharpened, is used. The cutting surface used is the end. This cutting surface glides over the tissue, making a clean, straight incision, not stretching the tissue, which may cause an irregular cut. If this is not possible on account of a large tonsillar mass, the tonsil is turned to expose the line, and it is severed by short incisions under direct vision. The incision is just through the mucous membrane and extends the full length of the tonsil and to the lowest level of our resection. About two-thirds of the way down, there will be noticed a fold or deeper attachment which is the posterior limit of the transverse fold. If this is not severed it may cause the snare wire to override the muscle or tear the membrane from the posterior pillar. This fold is the second of the three point suspension. As a rule, it constitutes the only point of difficult dissection along the posterior perpendicular half of the tonsil. It may or may not extend to the mucous membrane attachment, but if it is not correctly dissected there will be scarring in the fossa, including the posterior pillar.

The second incision is made just through the mucous membrane, following the anterior border of the anterior pillar, so that the entire mucous membrane over the plica triangularis is left adherent to the tonsil and removed. The incision extends the entire length of the plica triangularis. The two incisions are joined by severing only the mucous membrane attachment. The

dissection is started at the point of the second injection. A forceps or hook grasps the tonsil through the plica at this point. The same care should be used here as in the submucous resection. The mucous membrane only is dissected at first, but soon thickened strands of the palatoglossus with their aponeurosis covering will be encountered. The line of cleavage is between these two, and care should be exercised to remain in it. The combined hook and pillar retractor is of use here. Tunneling backward and upward the dissection proceeds as a dry dissection. No vessels are encountered until the perpendicular fold is reached. Following it upward we are led forward as it travels the crest of the gland. This leaves the dissection of upper pole complete except for this band. A hemostatic forceps in line of this fold grasping it is held in place while the gland is torn from it. This takes care of the superior tonsillar artery, which otherwise may cause trouble, as it is hard to find securely hidden in the vault. The separation of gland and ligament is completed by repetition of this procedure down over the back of the gland. The forceps acts as a hemostat to control the bleeding. Ligatures can easily be placed as we proceed, thus tying our vessels before they are severed, although with this new hemostatic forceps it is seldom necessary.

The dissection of the transverse ligament is now taken up. Its fibers arch backward and downward from a pointed start, spreading as they proceed. Sharp dissection should be used at the start, but as this is also a fibrous attachment blunt dissection is possible, although I prefer sharp dissection working against the capsule. This when completed usually frees the tonsil. It is often necessary to use the snare on the lower mass of lymphoid tissue and fascia which Barnes has termed the root of the tonsil.

CONCLUSION.

The attachment of the folds of the pharyngeal fascia to the gland marks the highway to a fossa lined with fascia, where there will be less bleeding, less infection and less scarring. They also surround the capsular and lymphoid tissue, all of which should be removed.

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Ross Hall Sullens

ROSS HALL SKILLERN, M. D.

1875—1930

Dr. Ross Hall Skillern, A. B., M. D., F. A. C. S., of Philadelphia, a member of the Editorial Board of the ANNALS, died September 20th, 1930, at the age of fifty-five.

Dr. Skillern, one of the outstanding contemporary laryngologists, was known especially for his textbook, "The Catarrhal and Suppurative Diseases of the Accessory Sinuses of the Nose," now in its fifth edition. His early education included Martin's School in West Philadelphia and the School of Biology of the University of Pennsylvania, from which he entered the Medical School of that University in 1893. After graduation he practiced with his father in West Philadelphia and began the special study of rhinology in Vienna and Göttingen in 1906.

He was Professor of Laryngology in the Medico-Chirurgical College from 1913 to 1919, and Professor of Laryngology in the Post-Graduate Medical School of the University of Pennsylvania from 1919 to the time of his death. He belonged to the Phi Gamma Delta fraternity and the following medical societies: The American College of Surgeons; the Philadelphia Laryngological Society, President 1912-1914; the American Laryngological, Rhinological and Otological Society, President 1929-1930; The American Laryngological Association, Vice President, 1925; American Academy of Ophthalmology and Otolaryngology, Vice President 1921-1922; the American Endoscopic Society; College of Physicians in Philadelphia; American Medical Association, Chairman of the Section on Laryngology, 1921.

During the World War he served as Commanding Officer of Base Hospital No. 89, A. E. F., at Mesves-sur-Loire.

He was a member of the National Board of Examiners in Otolaryngology, in which he was active from the time of its inception until his death. He was a prolific contributor to the literature of sinuology. A complete list of his work follows:

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Maxillary Sinusitis from the Dental Standpoint. Read by invitation before the Academy of Stomatology at the stated meeting, November 22, 1910. The Dental Brief, March, 1911.

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HENRY MORTIMER CUNNINGHAM, M. D.

1865—1930

Henry Mortimer Cunningham, M. D., Columbia University, 1895, died August 28th, aged 65, in his home, Vancouver, British Columbia, from a sudden kidney insufficiency. Dr. Cunningham was a Fellow of the American Laryngological, Rhinological and Otological Society and was Chairman of the Western Section two years ago; the Pacific Coast Oto-Ophthalmological Society and at one time its President. He was a Fellow of the American College of Surgeons and of the College of Physicians and Surgeons of British Columbia; a member of the Canadian Medical Society and the Vancouver Medical Society. He was one of the pioneers in bronchoscopy on the Pacific Coast.

Abstracts of Current Articles.

Surgical Treatment of Otosclerosis (Sur le Traitement Chirurgical de l'Otosclerose).

Prof. M. Sourdille (Nantes), Rev. de Lar. Ot. & Rhin., 51:341, May, 1930,

Presented by invitation before the otologic section of the Royal Society of Medicine in London, this important communication summarizes Prof. Sourdille's continuation of the work begun by Jenkins, Holmgren and Bárány. The operation is in two stages: (1) Atticotympanotomy, simple or with a plastic flap, excluding the drum cavity from the subsequent operative field, thus minimizing the possibility of labyrinthine invasion. Also ossicular deformations, adhesions and the like are set free and notable reduction of tinnitus may be secured by this prophylactic first step.

(2) Aseptic opening of the arch of the external semicircular canal, and covering of this opening by a thin epidermal membrane. This step must be performed by very delicate small special rasps, not roughly done by ordinary burrs. The membranous labyrinth must not be damaged, and the epidermal flap used to cover the opening is cut from the newly formed thin lining of the mastoid cavity. Subject to scarring, ulcerative destruction or keloidal thickening, this delicate membrane makes or mars the success of Sourdille's procedure. Thiersch grafts are too thick. Of his six cases, two have failed because of separation or ulceration of the epidermal graft following the first severe vertigo which lasts a few seconds. During the first week, while paraffin gauze is in place, hearing drops away down, but after edema has disappeared and smooth healing has occurred, hearing rises almost to the high level noted during operation.

Persistence of the bony opening may be tested by gentle compression after healing is complete, when the usual posterior fistula symptoms are manifest.

His oldest case has retained the improvement for a year. With perfected technic, Sourdille hopes to reopen the possibility of cure to a considerable group of progressive cases. Recovery of hearing seems to depend on five elements:

1. The previous degree of deafness;
2. Decompression of the perilymph;
3. Size of the artificial opening (larger the better);
4. Direct access of sound waves to the labyrinth via the new opening;
5. Both bone and air conduction are improved, especially for the lower range of sounds, including those of ordinary speech.

F.

Operative Technique for Chronic Progressive Deafness or Otosclerosis. (Nouvelles Techniques Opératoires pour le Traitment des Surdités Chroniques.)

Prof. M. Sourdille (Nantes), Arch. Int. Lar. 9:200 (Feb.) 1930.

This article gives accurate details and numerous pictures of Sourdille's method of opening the external semicircular canal with formation of a membranous flap over the fistula, as reviewed previously. F.

Calcium Therapy for Migraine (La Terapie Calcica Dell'emicrania).

Prof. V. Scimone (Padua), Minerva Medica, 21:859, May, 1930.

Considering migraine an allergic phenomenon, although he was unable to elicit skin reactions with the foods and pollens thought to be at fault, Scimone treated a series of sixty cases with intravenous injections of 10 per cent solutions of calcium chloride or gluconate. He uses twelve to sixteen injections of 10 cc. daily or on alternate days, and issues strictest warning against loss of the solution in attempting to locate the vein, because of its caustic action. He reports cure of all cases for periods from four months to two years; several recurrences were stopped by a second course of injections. Very slow injection is necessary on account of annoying subjective disturbances of taste and a feeling of warmth in the throat, mouth and other mucosal surfaces. Most of his cases had other allergic, asthmatic or eczematous findings. F.

Mineral Spring and Climatic Therapy in Otolaryngology. (Indications Creno-et climato Thérapiques en Oto-rhino-laryngologie).

H. Flurin (Cauterets) and J. Vialle (Nice), Arch. Int. Lar. 9:129, 257 (Feb. and March), 1930.

This important report, of 130 pages, was presented to the French Otorhinologic Society last October, and merits attention because so little systematic study of hydrotherapeutic establishments has been made in the various health resorts of America.

Speedy results from surgery, and inexact methods of saline and bath treatment, caused virtual abandonment of the latter during the past twenty years. Dangerous methods of douching and nasal irrigation were practiced by spa physicians and led to serious complications.

Two classes are suitable for such treatment: Chronic, slow, hyperplastic types, with abundant secretion, which react favorably to sulphur springs; and irritable, dry types which are subject to frequent acute infective or toxic processes and are helped by mild alkalin (sedative) waters. Arsenical springs are helpful in children with glandular enlargements, as well as for syphilitics.

The authors stress the descending nature of involvement of the respiratory system. Disapproving the crude and forcible methods of nasal douching formerly in vogue, they highly approve inhalation of steam from hot springs, also atomization of these waters. For chronic pharyngitis douching with a fine, hot stream directed through the mouth, as well as the hot coarse spray are found useful.

Sedative inhalations with mild alkalin waters are much in vogue for the chronic laryngitis of public speakers, but climate must be mild and free from wind.

Asthmatics, whose loss of sympathetic equilibrium is so frequently started by nasal anomalies, and hay fever sufferers, may be helped by the sedative watering places, but not cured. Desensitization and appropriate surgery must precede convalescence in such surroundings.

Hydrotherapy at mineral springs is absolutely contraindicated in cases of hypertension, cardiac incompensation, inveterate dys-

peptics, severe hepatic cases, the cancerous and especially the tuberculous.

Improvement of elimination by the purgative and diuretic action of mineral waters, baths, freedom from work and worry, and exercise going to and from the springs, are important factors in the cure of inveterate, discouraged cases.

Details regarding every important French and Swiss mineral establishment are included in the report.

Climatotherapy, depending on temperature, solar activity, humidity, prevailing wind, geologic and geographic factors, elevation and the like, is not as yet thoroughly systematized in France.

Mountain climate, powerfully stimulative, is most helpful for, but not curative of, laryngeal tuberculosis. Wind and fluctuating temperature are very harmful; and the lung condition, not the larynx, should decide whether "mountain therapy" is indicated. Lupus and tuberculosis otorrhea are helped by high altitude sun baths.

Functional insufficiency of nasal breathing—respiratory reeducation after adenoid removal—reshaping the deformed chest —are helped by high altitudes. Ozena, *per contra*, is better off at sea level.

Laryngismus stridulous, functional aphonia, labyrinthine anemia, as well as recurrent bronchitis and sinusitis, do well at mountain resorts. Convalescence from diphtheria, otitic septicemia, phlegm of the throat, as well as postoperative recovery after mastoid, larynx or sinus operations, is hastened under the influence of brilliant sunlight and great elevation.

Cachectic, aged persons or those suffering from acute purulent disorders—pyelitis, salpingitis; or the cardiovascular types, especially arteriosclerotics; or the active, febrile, congestive tuberculous; such people may not go to the mountains. Otosclerosis is unfavorably influenced, and both ozena and organic nasal insufficiency, unoperated septal deflections, adenoids, high palatal vaults, etc., are not helped.

Low levels—flat country with little wind and pleasant, sunny exposure—are helpful in the advancing and the congestive forms of tuberculosis. Such sedative regions are valuable also for the

neurotic victim of various paresthesias and hyperesthesias, as well as for otosclerosis.

The marine climate, if lacking in fog, is helpful (along the Mediterranean) in mild laryngeal phthisis, and even more so for nasal lupus. Atrophic and allergic rhinitis, spastic coughs of childhood, delayed postoperative healing, are helped by the sea. But the adenoid case will be made worse and require early operation. Acute suppurations of the ear or sinuses, hypertrophic rhinitis, chronic hyperplastic pharyngitis and laryngitis should seek higher ground; and exposed, windy sea beaches are a death warrant for tuberculous laryngitis.

The index, of French and Italian titles, covers thirty-four pages. F.

Remote Trepanation of the Skull for Otitic Complications. (Trepanation Cranienne a Distance.)

H. Aboulker and A. Badaroux (Algiers), Arch. Int. Lar. 9:385, April 1930.

The authors reject dural puncture through the mastoid wound and prefer to make a clean trephine opening through the squama temporalis, or the occipital bone, depending upon localizing signs. An extensive mastoid operation with exposure of the meninges should precede the trephining. The decompression alone will prove helpful even if pus is not reached by puncture. F.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

To better carry on its work, the American Board of Otolaryngology will increase the certificate fee from $25.00 to $50.00. The new rule becomes effective January 1, 1931.

An examination was held in Detroit, Mich., Monday, June 24, 1930, at which time seventy-eight candidates were examined, of which sixty-five were passed, one conditioned in pathology, and twelve failed.

The next examination will be held in Chicago, Monday, October 27, 1930. Those interested will please advise the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Neb.

H. P. Mosher, M. D., President.

W. P. Wherry, M. D., Secretary.

Society Proceedings.

ABSTRACT OF TRANSACTIONS OF THE AMERICAN OTOLOGICAL SOCIETY.

SIXTY-THIRD ANNUAL MEETING, MAY 20TH AND 21ST, 1930,

NEW OCEAN HOUSE, SWAMPSCOTT, MASS.

ABSTRACTED BY DR. ROBERT SONNENSCHEIN, CHICAGO.

TUESDAY MORNING SESSION, MAY 20, 1930.

The sixty-third annual meeting of the American Otological Society, Inc., convened at the New Ocean House, Swampscott, Mass., at 10:00 A. M., Dr. D. Harold Walker, vice-president of the Society, presiding.

Special Diet in Early Cases of Otosclerosis (Preliminary Report).

DR. EUGENE A. CROCKETT, Boston.

Owing to illness, Dr. Crockett was unable to read the above paper.

Calcium, Phosphorus and Cholesterol in Otosclerosis.

DR. EDMUND PRINCE FOWLER, New York.

Many observers have reported a diminution of blood calcium, and a few of phosphorus in otosclerosis.

The factors influencing absorption of blood calcium from the food are:

1. Amount of calcium in the food.
2. Acidity.
3. Available fatty acids for calcium soap formation.
4. Vitamin D, which in some obscure way increases calcium absorption.

The factors which influence the excretion of calcium are:

1. The slight calcium conservation in the body, because excretion goes on at a high minimum level under practically all conditions.

2. Excretion may be raised by an excess and lowered by an insufficiency in:

a. Acid (as in diets and acidosis).

b. Excessive parathyroid activity.

c. Excessive thyroid activity.

These factors favoring excretion are identical with those favoring the depletion of calcium from the bone, because the calcium in the bones is the only fixed basis easily available in time of need. Calcium phosphate is more soluble in the blood than in water because of the large proportion of protein in the serum, and it is less soluble in intracellular fluids because of the small proportion of proteins in the latter. It is therefore always potentially precipitable in the intracellular fluids where the proteins are low and the solution less stable. The most popular theories as to why it precipitates locally are:

1. The calcium already present precipitates from the supersaturated solution.

2. The removal of protein by certain cells precipitates the calcium.

3. Certain cells, especially cartilage, have a specific attraction for calcium.

4. Local change in pH. Under this head comes the influence of deficient circulation and diminished CO_2 formation.

5. That dead and autolysed bone is the source of calcium in fracture healing (Murray).

6. Enzymes are present which precipitate calcium-phosphorus by catalysis and by locally changing the pH (or both) (Robinson).

The last three of these are the more rational and do not conflict with one another or with observed phenomena.

A slow metabolism and therefore a diminished CO_2 production prevails in cartilage and other tissues prone to ossification. The pH in the tissue fluids in healing fractures is high (more alkalin) than the pH in the blood serum. Murray obtained increased speed in fracture repair and eliminated nonunion by the local planting of calcium salts. His results were probably dependent upon the local changes in pH.

Robinson's enzyme theory is the most plausible yet advanced, and since enzymes work best at a certain pH and often tend to change pH themselves, his theory fits in very nicely with the two

previous theories and supplies the local activator which was missing from them.

Fowler emphasizes the point that bone diseases can and do occur in spite of normal calcium, phosphorus and protein in the blood and that the direction and rate of flow are more important than the concentration in the latter, both calcium and phosphorus being very stable.

In fifty-eight cases of otosclerosis diagnosed by Fowler, using the usual criteria, the blood was examined for calcium, phosphorus and cholesterol by Dr. Andrew A. Eggston in the Manhattan Eye, Ear and Throat Hospital. The methods are given and the results graphically shown by chart in several age groups of the patients examined. In but three cases was the calcium below 9 mgms.; and in but seven above 11 mgms. per 100 cc. of plasma. For phosphorus only one was below 2.5 and two above 4.5 mgms. per 100 cc. The normal limits obtained under the conditions by examining 44 adults and children as controls showed the variations in calcium-phosphorus to be almost identical in the otosclerotics and the controls, considering also the seasonal variations. From every point of view, it appears certain that in established otosclerosis no change of consequence can be detected in the blood calcium-phosphorus variations.

The origin of cholesterol is unknown, but all body cells contain it. It is increased in several pathologic conditions, which have been associated with otosclerosis, especially pregnancy, early arteriosclerosis, hypertension, obesity, diabetes, chronic infections, alcoholism, syphilis, low basal metabolism and convalescence from pneumonia. It is diminished usually in febrile diseases, anemia and high basal metabolism. Experimentally degeneration in many parenchymatous structures may be brought about by increase in cholesterol and also a lessened phagocytic activity and antibody formation in animals. It is said to accelerate cancer growth.

Variable quantities of ergosterol occur in impure cholesterol. Irradiated ergosterol increases the calcium-phosphorus load of the blood and is efficient in the prevention and cure of rickets.

Fowler examined all of his cases for cholesterol and found few variations from normal. He concludes therefore that cholesterol shows no variations in established otosclerosis. These and

the foregoing concepts in the calcium-phosphorus problem are considered in detail and full credit is given to all authors. A very complete bibliography is appended to the paper.

Hearing in Children of Whom Both Parents Have Otosclerosis.

Dr. C. B. Davenport, Carnegie Institute of Washington.

This is a preliminary report of genetic studies made with the support of the Committee on Otosclerosis, with the aid, especially, of Drs. Duel, Harris and Dench, and with the co-operation of the Volta Bureau. It is based on data collected by Dr. Bess Milles on 100 families in which one or more members are affected by otosclerosis. The purpose of the whole investigation is to contribute toward the knowledge of the factors that are responsible for this defect, with special reference to the genetic factors.

Earlier studies made upon the genetic factors in otosclerosis have led to contradictory conclusions. It may well be that the clinical entity, otosclerosis, is not a genetic entity. There may be two or more genetic bases for a single group of symptoms that are classified clinically as otosclerosis.

The present is a progress report concerning the offspring of the mating of two parents, both affected by sclerosis. Seven pedigree charts were presented, including the selected mating. From the first mating of this type were derived eight children, seven otosclerotic. The remaining one died some years ago. He is believed not to have been deaf. The father of the fraternity had married earlier a normal woman, and of two sons who survived infancy one was in the Civil War and ascribes to heavy artillery fire a deafness to which he was subject. The other, who died many years ago, is believed to have been hard of hearing, but no details are available. The second pedigree is derived from Hammerschlag, who gives the offspring of an otosclerotic uncle with his otosclerotic niece. There were five daughters and two sons, all said to have been hard of hearing. In a third pedigree, derived from the Volta Bureau, a progressively deaf woman married a man who became progressively deaf after 45 years of age, and their only child, a daughter, has increasing deafness. A fourth family, with a mating of two otosclerotics, referred by Dr. Dench, has yielded only two offspring from the otosclerotic parents, both under 13 years of age—too young to show the symp-

toms. Three other pedigrees, including the mating of two otosclerotic parents, have yielded only otosclerotic offspring, with the exception of two children of 10 and 14 years.

It is a characteristic of simple recessive traits that when they show in both parents they appear in all of the offspring. These conditions are nearly fulfilled in the pedigrees presented. The most striking exception is the case of one child out of a fraternity of seven who alone remains unaffected. It was concluded that in the families presented the bony defect upon which otosclerosis depends was due to the absence of at least one genetic factor (there may be two) that makes for normal bone production and bone maintenance.

The Bone Picture of Otosclerosis and the Theory of Its Experimental Reproduction.

DR. MORITZ WEBER,

The George Williams Hooper Foundation for Medical Research, University of California, San Francisco, Calif.

The bone-forming system is composed of five so-called "time phases" of the normal development of bone. A combination within the body constitutes the "bone picture." A healthy individual has a "normal" bone picture; disturbances within the system result in a "pathologic" bone picture whose stages are controlled by the elements of time, space, quantity and quality.

The bone picture of otosclerosis as now well established is as follows: (1) Time: Postfetal (or fetal?). (2) Space: Localized, at certain areas of predilection within the labyrinthine capsule. (3) Quantity: Analogous to the blood picture, absolute displacement to the "left," caused by irritation (progression) and arrest of development (regression). (4) Quality: Faulty differentiation of the bone-forming system.

The bone picture of otosclerosis is identical with that of a localized osteodystrophia fibrosa ("osteodystrophia otosclerotic"). Both diseases may develop along the same course, but it is not essential for them to have the same pathogenesis in order to present the same end picture. The hyperplastic otosclerosis is a pseudotumor which represents the bone picture of "brown tumors." Theoretically, they are described as hamartoplasias.

The diagnosis "otosclerosis," from a histologic standpoint, in the presence of generalized osteodystrophia fibrosa, is only feasible in von Recklinghausen's disease (ostitis fibrosa with pseudotumors and cysts), and then only if the changes themselves within the labyrinthine capsule represent a typical pseudotumor.

A theory which aims at the reproduction of otosclerosis experimentally is based on the fact that this disease in man has been frequently observed simultaneously with generalized osteodystrophia fibrosa or with osteogenesis imperfecta (brittle bones). Two hypothetic methods are discussed by which the bone picture of otosclerosis might be reproduced. (1) Otosclerosis as a pseudotumor in generalized osteodystrophia fibrosa—i. e., as part of a von Recklinghausen's disease. (2) Otosclerosis as a pseudotumor in osteogenesis imperfecta (intrauterine scurvy?).

Reproduction of generalized osteodystrophia fibrosa has been experimentally induced in dogs through a purified diet low in calcium and completely lacking in vitamin D. In the jaw-bones typical "brown tumors" (intraosseous epulis) were observed. The labyrinthine capsule proper was involved in form of a generalized hypo-ostotic porotic form of osteodystrophia fibrosa. Lack of sufficient cartilaginous remnants at the time when the experiment was started is held responsible for the fact that genuine otosclerosis failed to develop. However, in the labyrinthine capsule anatomic changes were recorded which, although they were generalized, resembled the initial stages of otosclerosis.

DISCUSSION.

Dr. Theodore H. Bast, Madison, Wis.: I wish to thank the Committee on Research for the splendid help they have given me in the work I have been doing at the University of Wisconsin. I think Dr. Pierce has been overkind in his remarks. If it had not been for the financial help from this Society, which gave me an opportunity of getting a very good assistant, the work would never have been done.

In connection with my work, I would like to make one appeal, if possible. I have studied the normal developmental aspect of the petrous capsule, and I am anxious to get fetal material coming from parents who are hard of hearing. It may be possible, from

time to time, to get a fetus from such a source. A half term or full term fetus, or in the early stages after birth would be of great value. If any of you can help in obtaining this material I would appreciate it. These petrous bones can be preserved in a 15 per cent formalin solution—that is, 15 parts formalin in 85 parts of water and will keep for weeks at a time.

Dr. Weber's work has interested me very much, not that his ideas are not entirely in agreement with mine (and unfortunately or maybe fortunately, very few workers are in agreement on the question of petrous bone pathology), but because his papers have been thought-provoking. In the second place, I have been interested because Dr. Weber has given us in this and his previous work a comprehensive review of the opinions of the German workers, together with his own courageous interpretation of the histologic picture of petrous bone pathology.

With the permission of the Society, I should like to comment on several structures to which Dr. Weber has referred.

1. So-called web-bone of Max Meyer or fiber bone and lamellar or shell bone as related to (a) normal types of bone, and (b) pathologic types of bone.

Dr. Weber has mentioned time, place, quantity and quality of bone formation. There is a fifth factor, namely, speed of bone formation, which is very important in determining whether the finished bone is a so-called web bone or a lamellar bone. The so-called web bone which is found in primary endochondral bone, membrane bone and rapidly forming bone, such as neoplasms, are dependent largely upon the rapidity with which they are formed, and in neoplasms may be augmented by the degree of inflammation.

The cells of this type of bone are less highly differentiated cells than those which form lamellar bone and are capable of laying down intercellular connective fibers, amongst which they become enmeshed before they become true endoblasts and lay down bone. In short, web bone is formed very rapidly by very active and rapidly metamorphosing cells. The lamellar bone is much slower in formation and the cells which form it are more highly specialized osteoblasts before they begin their bone forming activity.

In a word, the slower the formation of bone the more finely laminated it is, and the reverse, the faster the process the more weblike the bone becomes. One can under different conditions or locations see all the intermediate stages between the so-called web bone and highly developed shell bone. The difference is only one of degree and not one of type.

What application has this to bone pathology? Dr. Weber (if I understand him rightly) stated that shell bone does not occur in the otosclerotic patches, but only weblike bone similar to that of ostitis fibrosa. I have recently, in looking over Dr. Wilson's preparations, seen localized otosclerotic patches in which the new formed bone was lamellar, and where the remaining bone marrow was not highly fibrous. Now if one lays great emphasis upon web bone and lamellar bone as distinct types, then one would have to consider the lamellar otosclerotic patch as a distinct pathologic bone picture. My interpretation is that all these localized otosclerotic patches are of one type but differ in degree due to a difference in the speed of development. The only essential difference in type that I can conceive is one in which inflammation or nutrition has augmented the degree of fibrosis.

2. The second point which I wish to discuss is the method of resorption. I was interested in Dr. Weber's statement that bone resorption is accomplished by either osteoclasts or blood vessels. Resorption of bone by osteoclasts is of course well recognized, but I have not observed direct resorption by blood vessels. I would like to ask Dr. Weber where and how this occurs. If we take the most recent concept of osteoclasts as a fusion product of connective tissue monocytes (mononuclear phagocytes), then we must remember that in cases where osteoclasts as such are not seen it is still possible for these monocytes to absorb bone without being fused into osteoclasts.

I agree with the essayist that bone is not resorbed as long as it is functionally alive, but only after degeneration has set in, due to one or the other of various causes. Blood vessel pressure can produce this degeneration as may be seen in Fig. 1. Here, due to the growth of bone, the blood vessel in the nutrient foramen presses against the bone on one side, and here osteoclasts are seen removing bone. On the other side, where there is no such press-

ure, the osteoblasts are actively laying down bone. The same condition exists in Fig. 2, where a branch of the inferior dental nerve passes through the mental foramen. Here the nerve instead of a vessel causes pressure, and the large osteoclasts can be seen removing bone, while, on the other side osteoblasts build bone. This pressure in either case, I believe, causes damage to the bone and results in its removal by osteoclasts and not by blood vessels direct.

3. The third subject which calls for comment is: Are the so-called cartilage islands predilected areas for bone resorption?

O. Mayer thinks they are. Wittmaack thinks resorption occurs where there is venous stasis. Our essayist takes an intermediate view, namely, that vascular disturbances occur most easily in the region of these so-called cartilage islands.

I hesitate to agree with the view which many hold, that these islands are malformed bone. We must distinguish between two types of cells in these islands. In my previous articles on the development of the otic capsule, I have stated that the so-called cartilage islands consisted of intracartilaginous bone in a matrix of calcified cartilage. That is, bone cells invaded the so-called cartilage lacunæ. The cartilage cells are removed and these invading osteoclasts or bone cells lay down bone. This I feel is a normal process for the otic capsule and not a malformed bone. In Fig. 3 this normal process is shown in a 5 months' fetus. Here each old cartilage lacuna is shown with a nest of bone cells in each, instead of a single cartilage cell which occupied such space previously.

In Fig. 4, however, taken from the otic capsule of a fetus of about 5 months, the condition is different. In the light areas typical cartilage cells are still present, with only one cell in a lacuna. Around these light cartilage masses other dark bodies are seen. These are the same as in Fig. 3. They consist of two or more bone cells with their surrounding bone matrix occupying the lacuna which was previously occupied by a single cartilage cell. In Fig. 4, then, we have an imperfectly formed intracartilaginous bone with typical cartilage cells still present. In most of my preparations of a little later stage these are being removed by the invasion of osteogenetic buds, but in some cases

small areas of this type may persist. Such areas I feel would be readily subjected to later pathology. In the region of the fissula ante fenestrum typical cartilage cells persist quite regularly, even into old age.

4. The fourth and last point is: Are there any animals which have the so-called cartilage islands in the petrous bone to make them sufficiently similar to man to be used for experimental production of otosclerosis?

I think Dr. Weber is very brave in presenting an hypothesis as a basis for experimental production of otosclerosis. I have no comment to make on this part of his paper except to wish him God-speed in his endeavors. Dr. Weber certainly is an optimist, judging from his statements on experimental production of otosclerosis. Now if optimism begets results, I am going to present a fact which should increase his optimism and insure success. Here is the fact: So-called cartilage islands actually exist in some laboratory animals.

In most cases the condition is as shown in Fig. 5, a section through the otic capsule of the adult rabbit. Here the primary bone is shown by the darker areas, while the lighter areas are Haversian systems which have replaced the cartilage remnants. In a two-day old cat remnants of cartilage (black masses) are being removed by osteogenetic buds. In a three-week-old dog there is still considerable cartilage left in the otic capsule. Such animals, while still quite young, might make suitable material for experimental purposes.

The animal, however, which shows a condition which in part can be compared with man is the monkey. This animal, while most of its capsule is bony, has a cartilage area in the region occupied by the fissula ante fenestrum in man. (A true fissula like that in man has not been found in any of the laboratory animals.) Since it is this region of the capsule where the otosclerotic patches are most common the suggestion is most natural that the monkey may be suitable for experimental purposes. I hope that this information may be of some value to Dr. Weber in his endeavors at experimental production of otosclerosis.

Dr. Alfred Lewy, Chicago: Dr. Fowler in his paper refers to calcium, phosphorus and cholesterol in otosclerosis, and men-

tioned the relation of calcium to phosphorus. I would like to call attention to the fact that fluorine also appears to have some effect in bone metabolism. There is considerable literature on this subject. Among others, McCallum of Baltimore showed the influence of minute doses of fluorine on the growth of the teeth and skull bones in rats. At the Washington meeting of the Triological Society in 1928, Dr. Roy Barlow presented specimens of skulls and teeth of rats showing changes apparently produced by fluorine feeding. At the same meeting I presented slides showing what appeared to be changes in the bony labyrinth of mice which had been fed with fluorine. At that time I called attention to the fact that the bone took the hematoxylin stain more readily and that the lacunæ were larger and more frequent than in the controls, which I interpreted at that time as the appearance of immature bone. Since then I have submitted my slides to Dr. Joseph Fisher, histopathologist at the Alexander Clinic in Vienna, and he pointed out to me in one specimen a deposit of osteoid tissue on the footplates of the stapes and on the neighboring labyrinthine wall.

I mention this now to bring it to the attention of the Committee on Otosclerosis, in the hope that they will have this subject pursued further by a trained investigator instead of leaving it to a mere clinician like myself.

Dr. Thomas J. Harris, New York: Referring to Dr. Davenport's paper, let me say that I was greaty interested in his conclusions. So far as I know, this is the most complete paper on the subject which has appeared in American literature. To what he said I cannot presume to take exception. I would like to highly commend Dr. Milles' work in securing the data for Dr. Davenport's charts. The speaker has called attention to the relationship of heredity to otosclerosis. There can be no possible question on this point, for it has been studied very extensively. Therefore, in my experience, based on two years' work at the Manhattan Eye and Ear Hospital, the results came as a surprise to me. In the literature, Fraser, Gray and others cite a percentage of patients with clear heredity in the history, as high as 60 to 70 per cent. Our figures show a surprising difference. Dr. Milles did the work with extreme care, but in place of the 60 to 70 per cent of the

English workers, we were able to get only 8 or 10 per cent. If the work had not been done by a well trained worker I should have thought the error was in the methods employed. I have asked myself whether the statistics of the European investigators were collected as carefully—whether the statement "father was deaf" or "parents were deaf," was used to describe merely a suppurative condition. In less than 100 cases of otosclerosis we had no such percentage as the German and French authors quote. This has nothing to do with the question of the importance of heredity, but we found case after case which seemed to us genuine otosclerosis, where we could get no heredity in the history at all.

Dr. J. Gordon Wilson, Chicago: We have listened with great interest to this presentation of Dr Weber, and we congratulate him on the study he is making of the bone pathology of the ear. We would also send our appreciation of the work being done on the ear at the Hooper Institute, to our friend, Dr. Coleman, through whose generosity and stimulation this work has largely been made possible.

Changes in the petrous bone are by no means uncommon in disease, but the relationship of this to otosclerosis is hard to determine. The end pictures are so different that I feel that the pathogenesis cannot be altogether similar. The relationship of osteodystrophia fibrosa (ostitis deformans) to otosclerosis, though showing much similarity, also shows great dissimilarity. Last summer I had an opportunity of seeing some of Dr. Jenkins' specimens. The relationship of the disease to otosclerosis has specially engaged his attention. His specimens show, and his opinion is, that the disease (though pathologically very much alike) are nevertheless in their pathogenesis very different.

The pathologic picture one sees in otosclerosis is by no means always the same. In the region of the stapes-vestibular joint my specimens show two outstanding types: (1) One resulting in dense bone sharply outlined from the surrounding tissue, with no apparent change in the periosteum or endosteum; and (2) the other type which is distinctly osteoperotic, and associated with surrounding disease in the mucous membrane of the middle ear.

Both are localized and both result in stapes ankylosis. It is hard to believe that they are the result of identical factors.

Dr. M. A. Goldstein, St. Louis: I think Prof. Davenport would revel in the story of an otosclerotic family concerning which I am now collecting data. There is a history of otosclerosis in five generations, four of which are living and three of which I have seen. In these three present generations there are eleven members in the family, and from the clinical facts and functional test reports that I have been able to gather, I feel we are justified in diagnosing this entire group as clinical otosclerosis. One of the most interesting observations that occurred in connection with this family is that of a three-year-old child of the last generation whom I have determined to be congenitally deaf.

These sequences of hereditary transmissions may not be in accord with Mendelian ideas, but it may develop new angles in the hereditary analysis of such a group, and it should also stimulate further investigations as to the possible association of otosclerosis and congenital deafness.

I wish to emphasize one point to which Professor Bast referred and which I think should be given serious consideration in the further investigation and study of otosclerosis in various aged embryos where clinical otosclerosis has been previously diagnosed in the mother.

Two months ago I was consulted by one of our outstanding obstetricians in the case of a young woman of 21 in whom I had diagnosed definite clinical otosclerosis at 17 years of age. Her hearing had been rapidly growing worse; she was pregnant, and I was asked what effect pregnancy and childbirth had on this type of progressive deafness. It has been a well known observation by many otologists, verified by my personal experience, that every childbirth in an otosclerotic predisposes to a further depreciation of the hearing.

Under the circumstances and in this particular instance, where the hearing was getting rapidly worse in both ears, abortion was agreed on as a justifiable measure to safeguard the mother. In this case a three-months fetus was secured.

No literature could be found on the question of examination of embryos in otosclerotic mothers. Professor Bast and Professor

Streeter were consulted about such literature, but they were unable to supply it.

I think this is a valuable angle from which to study the question not only of bone formation, but also of heredity. If we can get embryos, at different ages, of otosclerotic origin, it seems to me it would be important material with which to work.

Dr. Edmund P. Fowler, New York: I should like to remark that one should be very careful in saying that all these cases who have had either deaf children or a history of deafness in the family are otosclerotics. If we chart out deafness aside from otosclerosis we often get family trees showing deafness in the offspring very similar to that in otosclerosis. Like tuberculosis, most forms of deafness are not inherited, but may be acquired through certain anatomic and physiologic peculiarities which are inherited. There may also be undue exposure to or a tendency to infection, as in tuberculosis. Many inflammations (unrecognized in children, and which may be denied, yet with evident scars) besides otosclerosis may cause progressive deafness, but it is often impossible to differentiate them from otosclerosis. Therefore we must be very careful in making a diagnosis of otosclerosis. I should like to mention a little trick I have discovered to aid in making the diagnosis of otosclerosis. "Bezold's triad" and the "Schwartze symptom," the pinkish glow of the promontory as seen through the drum membrane, are the only known phenomena of importance in the diagnosis of otosclerosis. Unfortunately, negative Rinne or so-called increased bone conduction with loss of hearing for the lower tones and progressively better hearing as we ascend the tonal scale and for the Galton whistle may all be found in cases of obstructive deafness other than otosclerosis; and there is usually no way to differentiate between the two. One is particularly helpless in differentiating cases with slight loss of hearing. Excluding middle ear and labyrinth inflammation, when hyperemia in the mucosa is indicated by a reddish glow of the promontory, a diagnosis of otosclerosis is warranted. Unfortunately, in a large proportion of cases otherwise typical of otosclerosis, one is unable to detect any congestion on the promontory. I have contrived a method for a better vision founded upon a phenomenon which has stared me in the face for twenty

years without significant recognition. This phenomenon is the visibility of congestion areas upon the promontory, which though invisible with the ordinary methods of examination, become apparent by slowly stretching and approximating the drum membrane to the promontory by means of a magnifying focusing pneumatic speculum. To better evaluate color variations, a daylight lamp is used in this connection, and for that matter, in all my otoscopic examinations. It is not difficult to surmise why this simple procedure should elicit the phenomenon in question, because when the drum membrane is stretched by the positive pneumatic pressure, three things take place:

1. The drum is slightly thinned, which makes it more transparent.

2. Its light reflexes are changed so that the background for comparison is altered.

3. The color and visibility of a colored object viewed through a semitransparent membrane increases as the membrane approaches the object. (In other words, the red color becomes more and more apparent as the drum nears the congested promontory.)

Some practice is necessary in interpreting any color changes, but with careful observation there should be little difficulty in the matter. Naturally visibility is aided by transparent and movable, and hindered by opaque and immovable drum membranes. The method is also useful in reverse, namely, to diminish a reddish glow as the drum is slowly sucked outward. If the red is on the promontory, it will fade as the drum recedes from it. If an increased redness appears during the negative pressure, one should suspect that the increased congestion is upon or in the drum structures. I have never observed this in uncomplicated otosclerosis.

The method I have described is particularly useful in children, probably because of the greater transparency of the tympanic membrane in the young. It is therefore of value not only for the earlier diagnosis of otosclerosis but for the diagnosis of early otosclerosis.

Dr. Moritz Weber, San Francisco (closing): Dr. Bast has mentioned the factor of speed. Speed is a function of time and space, hence it is included already in my definition of the bone

picture. Furthermore, I would like to stress another point: In the work of Weidenreich especially we have learned to distinguish two distinct types of bone in the fibrous bone, and in the lamellar or shell bone. While the first type is phylogenetically and entogenetically the older, the lamellar bone is the more recent. The latter is formed by, or in the presence of osteoblasts in epitheloid arrangement, while the former originates without cells arranged in a row. The fibrous bone acts as a center for the subsequent deposit of the lamellar bone. The lamellar-free bone, first described by Max Meyer, also represents a bone structure which is formed in the presence of osteoblasts in an epitheloid row.

Since examination of sections, as well as the literature, reveals that a typical lamellar bone, or bone originating in the presence of osteoblasts in epitheloid arrangement, is absent in otosclerosis, if one should find such bone in typical otosclerotic foci it would rather indicate a healing process. Osteodystrophia fibrosa, also, in its sclerotic stage, shows lamellar bone (see Christeller).

As to the question of bone resorption directly by blood vessels (without giant cells), I refer to the classical work of Tommer, mentioned in the bibliography of my papers.

By offering my conception of the bone forming system, I have attempted to present a basis for interpretation of pathologic conditions, including otosclerosis. I feel much obliged to Dr. Bast for discussing my paper, and I was indeed very much interested to see his slides in which he demonstrated a large cartilaginous remnant in a monkey at a place where the fissula ante fenestrum is seen in man.

In answer to Dr. Gordon Wilson, I wish to emphasize that only the histopathologic examination can give exact proof as to whether one is dealing with a case of otosclerosis or not. Therefore all studies of heredity in relation to otosclerosis will be of much greater scientific value for the problem of otosclerosis when the clinical observations can be confirmed by histopathologic diagnosis. Clinically it is more correct to speak of "progressive deafness," with suspicion of otosclerosis.

If I understand Dr. Norval Pierce aright, he does not believe that any dystrophy causes otosclerosis. In this connection let me

remind you of the fact that carcinoma is a tumor, and that carcinoma was produced first by Yamagiva by application of tar to the back of mice. Patients suffering from carcinoma of the stomach, however, never had tar applied to the abdomen. The question is, which common factor or factors cause the picture of carcinoma experimentally in mice and spontaneously in man? From the standpoint of experimental research we face a similar problem in regard to otosclerosis. I did not state that otosclerosis in humans necessarily originates from a dystrophy. But if research succeeds in producing the bone picture of otosclerosis by any means, excellent leads would be found to the understanding of its etiology in man. The subject of my paper was to present a workable hypothesis for the experimental reproduction of the bone picture of otosclerosis by means of a dystrophy.

Afternoon Session, Tuesday, May 20, 1930.

Gradenigo's Syndrome—Anatomic Aspects.

Dr. Philip E. Meltzer, Boston: The Gradenigo syndrome is attributed pathologico-anatomically to extension of inflammation from the apical cells of the petrous pyramid to the sixth nerve and semilunar (Gasserian) ganglion. Many excellent contributions have appeared since Gradenigo's original work, which would indicate that there are other pathways for the inflammation or toxins to be diffused to the region of the tip, and also quite capable of producing the syndrome.

There are numerous canals extending from the middle ear which transmit vessels and nerves which communicate with regions of the tip, either directly or indirectly. These vessels and nerves are connected by a fine, loose connective tissue, the perineural and perivascular tissue. Papale proposes the idea that it is through these perilymphatics that the inflammation or toxins are carried to the region of the tip, where the carotid artery and sixth nerve are in intimate relationship, and that the nerve is involved at this point. A diffuse inflammatory edema in the cells of this region has also been suggested as the cause.

Gradenigo assumes a Gasserian ganglion involvement in accounting for the severe pain which is present. This hypothesis can be objected to on the basis that the pain need not be gan-

glionic in origin. It may be due to the pressure within the cells, giving rise to pain, or due to pressure upon the nerves of the tympanum, and this may be referred to the regions which are commonly localized in this syndrome. The nerve connections of the fifth nerve with the nerves of the middle ear are quite definite, particularly through the otic and sphenopalatine (Meckel's) ganglion. Inflammatory or toxic involvement of these nerves could send impulses along these connections to the localized areas described by Gradenigo.

It seems reasonable to assume that in view of the recent pathologico-anatomic contributions to the study of this peculiar triad of symptoms, the Gradenigo syndrome may be due to: (a) Cells extending to this apex (paralabyrinthine, subarcuate, paratubal); (b) the carotid canal, (1) caroticotympanic canals, canaliculus tympanici, (2) by erosion of the bony wall of the eustachian tube; (c) the perineural and perivascular lymphatics of Papale; (d) erosion of tegmen tympani with extension forward; (e) inferior and superior petrosal sinus.

Osteitis of the Petrous Pyramid of the Temporal Bone Associated With Paralysis of the External Rectus.

Isadore Friesner, M. D., and J. G. Druss, M. D., New York: Whatever may be the cause of these differences, there are wide variations in the number, extent and size of the cells in the temporal bone. As a general rule, the larger the cells, the greater the facility with which infections spread through them. In the highly pneumatized processes it is not an unusual operative finding to uncover purulent disease in cells remote from the antrum. It is not clear why infections in the mastoid vary in their predilections for certain pathways. It is particularly true of the extensively large celled mastoids that the pneumatic structure extends about the bony labyrinthine capsule into the depths of the petrous pyramid. Just as the cells in the mastoid vary in number, extent and size, so too do the cells of the petrosa; and furthermore, just as in the mastoid an infection at times exercises a predilection for certain pathways, so a similar condition may exist in the pyramid. In the instances of acute middle ear infection associated with fifth or sixth nerve involvement due to an osteitis, which came under the author's observation, all had either

operative or X-ray proof of the pneumatization of the mastoid processes. It is conceivable, however, that even when the mastoid process is sclerosed, the pneumatic structure may yet exist in the petrosa. Many paths or routes of extension from the middle ear and mastoid cavities to the apex have been outlined, such as:

1. Sublabyrinthine route extending from tympanum below the labyrinth and internal auditory nerve to the petrous tip.

2. From the mastoid antrum through the subarcuate fossa, or the petromastoid canal, which passes inward beneath the superior semicircular canal and reaches the layer of cells above the internal auditory meatus to the petrous tip.

3. Carotid canal, by eroding the bone on the anterior tympanic wall or through one of the caroticotympanic foramina which give passage to the carotid branches of the tympanic plexus.

4. Layer of cells extending along the eustachian tube from the tympanum to the petrous tip.

5. Along the superior or inferior petrosal sinus to the apex.

6. Extension of an extradural abscess of the petrosa to the apex.

7. Indirect trauma at operation or otherwise.

All infections in the petrous pyramid do not necessarily extend to the apex.

There is still some question as to the exact nature of the pathologic process which occurs when the infection, having traveled through the pyramid, reaches the apex. The fifth nerve is undoubtedly involved at its ganglion and the sixth nerve in all probability in its course through Dorello's canal. The author's sections show definitely a periganglionitis and a ganglionitis.

Among other theories which have been advanced to explain particularly the involvement of the sixth nerve are:

1. As a labyrinthine reflex.

2. Toxic influences, such as rheumatic fever, influenza, diphtheria, etc.

The author's conclusions are:

1. Osteitis of the petrous bone as a causative factor in the production of adducent paralysis is much more prevalent than is believed. This may not always be demonstrable macroscopically but may be ascertained by histologic study.

2. The diagnosis of osteitis of the apex of the petrous pyramid can be correctly made in many cases.

3. The infection may subside in the tympanum and mastoid cavity, but may progress towards the apex of the petrous pyramid with increased severity.

4. The infection may extend through the bony substance of the petrous pyramid and reach the apex, or may perforate the cortex and involve the dura at any point in its course and then spread to the apex.

5. Anatomic variations of the petrous pyramids play an important rôle in the spread of infection in that structure. In the cases which recover spontaneously the infection is probably confined to the bone.

6. Inasmuch as there is no single anatomic pathologic basis for the so-called Gradenigo syndrome, the value of the term is doubtful.

7. Surgical approach to the apex of the petrous pyramid is indicated in the presence of progressive meningeal signs.

DISCUSSION.

Dr. George E. Shambaugh, Chicago: It is instructive to have the clinical phenomena of Gradenigo's syndrome presented with the anatomic and pathologic considerations such as we have listened to this afternoon in the two papers by Dr. Meltzer and Dr. Friesner.

Paralysis of the external rectus is not such an uncommon phenomenon when we include its occurrence with meningitis. As a symptom separate from other intracranial complications it is not very common. I am inclined to feel that the syndrome might be more properly described by some term such as paralysis of the external rectus occurring in acute otitis media. Pain may be conspicuous or it may be entirely absent. It does not constitute an essential part of the syndrome. Paralysis of the external rectus and the presence of an acute otitis media are the two essential phenomena.

How does the infection get through the middle ear to involve the sixth nerve? Dr. Friesner brought out very clearly that there

are several routes and that it is not always dependent upon infection of pneumatic spaces at the apex of the petrous bone. In my anatomic studies I have not been impressed with the frequency with which pneumatic spaces occur in this region. As Dr. Friesner shows, it seems more likely that the lymphatics about the carotid canal constitute the most frequent means for carrying infection forward so that alterations may be produced in the sixth nerve.

I have encountered several conditions where the paralysis of the sixth nerve constituted the conspicuous symptom. One I recall was a young man who had an ordinary acute otitis media, no evidence of a mastoiditis, nor was there any complaint of pain. External rectus paralysis and the acute otitis media were the outstanding symptoms. He made a spontaneous recovery—that is, without operation. Only recently I saw a child, not over 12, with an acute otitis media and paralysis of the external rectus, which also cleared up with no further operative treatment than the incision of the drum membrane.

Another case was a young nurse who previously had had a simple mastoid operation somewhere in connection with an acute otitis media. The mastoid healed and to all appearance there was complete recovery. She came to see me because of severe pain in the temporal region. Careful watching over two or three weeks failed to discover anything except occasionally elevation of temperature to 99. One day paralysis of the external rectus developed. The otitis media had entirely healed and the perforation in the drum membrane closed. An operation was undertaken with the idea of working forward toward the apex of the temporal bone. In the course of this operation an extradural abscess the size of a large pea was exposed anterior to the tegmen covering the attic—that is, the root of the zygoma. She made a rapid, complete recovery.

Another case was that of a man who was referred to me because of profuse, purulent discharge which had persisted for a couple of months after an acute otitis media. This was before the use of skiagraphs. Mastoid exenteration was carried out, but we

failed to discover the abscessed cavity which the profuse discharge led me to believe was probably there. He recovered rapidly from the mastoid operation, but the profuse discharge from the tympanum persisted. Because of this a second operation was undertaken, believing that the trouble had its origin in the cells leading off from the tympanum or the tube. Exploration of the hypotympanic space uncovered nothing in this region, but the pus could be seen oozing out of the tympanic orifice of the eustachian tube evidently from infection of a large tubal cell. With great care I undertook, with curettes, to open out this region, but succeeded in nicking the internal carotid. The bleeding was quickly controlled by a firm iodoform gauze pack which was ready for such an emergency. The next morning the patient was found to have an external rectus paralysis on that side. We had no further trouble with bleeding and the ear healed, with complete cessation of discharge but the external rectus paralysis persisted for something over a year before it entirely disappeared.

The problem that presents itself in these cases is the question of what we should do. I concur with Dr. Friesner in the belief that we should not hastily reach the conclusion that because the external rectus is paralyzed there exists some intracranial complication that requires surgical relief. If the patient has a mastoiditis, this should be an additional reason why the operation should be carried out, or if there are symptoms of a threatened meningitis the operation should not be delayed. But, all in all, with cases as they come, the chances are just about as good if we do not operate as if we proceed with surgical measures.

Dr. R. A. Fenton, Portland, Ore.: With reference to X-ray findings in these cases, the usual position taken is the lateral one. This was discussed at the Copenhagen congress, where Prof. Perussia of Milan suggested taking the X-ray from chin to vertex to determine the relative shadow of the involved side. The progress was noted by taking a succession of plates. We recently had a boy of seven under observation for four months who maintained a high temperature during most of this time. He had rectus paralysis with remissions and has gradually recovered. The chin to vertex position showed distinct darkening on the

paralyzed side and petrous pneumatization of both sides. This position for filming helped a great deal in determining the extent of involvement.

Dr. F. T. Hill, Waterville, Me.: A recent case has shown an interesting coincidence. The case developed Gradenigo's syndrome three weeks after operation on a very pneumatic mastoid. The middle ear was dry for three days, and then there was a profuse discharge. The temperature prior to operation and afterwards was 99 to 100. After the development of the Gradenigo's syndrome we reopened through the zygomatic region, uncovered the dura widely and found a small epidural abscess. The middle ear became dry but the temperature rose and stayed at about 102 to 103 for several days. Because of a possible exposure to undulant fever we examined the blood. It gave a positive test for para-typhoid A, confirmed by test on the stools. With proper treatment the patient made an uneventful recovery. The undulant fever, no doubt, accounted for the temperature.

Drs. H. P. Mosher and L. A. Schall, Boston: The autopsy findings in cases of Gradenigo's syndrome are pertinent to the subject under discussion. In a girl of nine, a left mastoidectomy was done March 14. The cortex was very thick, there was necrosis with pus around the antrum, and a deep layer of cells in front of the sinus. On March 20th, I tied the jugular; on the 31st, a zygomatic abscess was opened; on April 14th, she developed a left sixth nerve paralysis with pain deep in the left eye. The next day she vomited and continued to do so. About the 17th, the pain disappeared from the left eye but appeared in the right eye. Neurologic findings, other than sixth nerve paralysis, were negative. On April 30th, after vomiting, she passed into coma and died six hours later from respiratory paralysis. The autopsy findings are interesting and I quote the pathologist: "Upon removing the skull cap, the surface of the brain is found to be markedly edematous, the fluid standing out beneath the pia in a gelatinous layer. Upon removing the brain from the cranial vault an area of hemorrhage and softening is found on the under surface of the pons and medulla. This is 4 cm. in length, 2 cm. in diameter, 1 cm. in depth, and is intimately associated with the brain tissue lying about it. This hemorrhage mass

is covered with a fibrous layer. Smears show numerous neutrophiles and many gram positive cocci, many of which are growing in chains from four to six. Upon sectioning the pons and medulla, the substance of the brain is found to be very soft and infiltrated with fresh hemorrhage.

"At the point of junction of the sphenoid, temporal and occipital bones the osseous tissue is found to be necrotic and to contain between one and two cc. of purulent material. Over this area the dura is boggy, and upon the left posterior surface of the basilar process of the occipital bone the dura is raised and white, though no purulent material is obtained when opened at this point."

I believe that in this case the route of infection was directly through the petrous to the tip, in spite of an early and complete mastoidectomy.

Dr. Meltzer (closing): I have tried to discuss the symptoms of the Gradenigo syndrome from an anatomic basis, and I repeat that when we speak of Gradenigo's syndrome we necessarily must consider the triad of symptoms existing together, but quite agree that this term should be changed. I think we can have involvement of the sixth nerve without the fifth, and conversely, involvement of the fifth without the sixth. Of course, the primary factor must be a suppurative otitis media. The term Gradenigo's syndrome is used too loosely in the literature. I certainly would approve of a descriptive term which would include the symptoms, even though not necessarily including the fifth and sixth nerves at the same time.

Dr. Friesner, New York (closing): I am in agreement with Dr. Shambaugh's attitude on surgical intervention as regards the mastoid in these cases. I have seen several such instances: one recently in a girl of 12, following acute otitis, with no evidence of intracranial involvement, who had external rectus palsy. Some surgeons believe that this alone is an indication for mastoidectomy. With that I do not agree. I am interested in the case report of the nurse mentioned by Dr. Shambaugh, which emphasizes one point. In the presence of external rectus palsy, where there are menacing signs, I think one should look for trouble. The epidural abscess Dr. Shambaugh found was perhaps an extension from the cells of the petrous pyramid, as I have shown histologically. Two

winters ago I saw, with Dr. Richards, a young man of 17, with severe pain, profuse discharge, high temperature and extreme tenderness. At the operation the mastoid cells were found full of pus but without necrosis. The middle ear dried up, but ten days after the operation it discharged again, following the apparent resolution. I saw him at the time and found the boy looked gravely ill, but with no meningeal signs. There was a slight homolateral facial paralysis but very little fever. Dr. Richards reopened the mastoid and we looked into the depth of the wound into the antrum. It was covered with normal healthy granulations with no secretion, but in the very middle ear one saw a profuse discharge of a pale creamy color coming from a very much inflamed tympanum. There was no tenderness about the wound. I believed there must be a collection of pus somewhere in direct connection with the middle ear, probably in the petrous pyramid. Dr. Richards decided to sacrifice the middle ear. In the tympanic recess, in the same situation which Dr. Shambaugh described, there was a granuloma. A probe entered the pyramid and pus issued from the tract. Dr. Richards entered the petrous pyramid, removed the inner table and broke down the gutter behind the carotid artery in front of the facial nerve. The opening discharged about 2 cc. of pus. The patient died. I have attempted this procedure. Once I injured the carotid artery and spent some very uneasy moments but no fatality resulted. I think it has been borne in on me by my clinical experience that we otologic surgeons are dealing with two pneumatic processes readily infected. The first, the mastoid, is easily accessible; but the second, the pyramid, sometimes requires exploration. It is difficult to enter the petrous pyramid but not impossible. When there are meningeal signs and involvement of the sixth nerve the attempt is worth while, especially when one ear alone is suppurating. We have to sacrifice the ear and try to relieve the suppurating focus in the pyramid. If I awakened you gentlemen to the consciousness that something should be done to develop a technic to open the pyramid safely, that something should be done to prevent extension of the lesion in the petrous pyramid to the intracranial contents, I shall have accomplished the purpose of this presentation.

Five Years' Study of the Relation of Infection of the Ear and Infection of the Intestinal Tract in Infants.

DR. D. E. STAUNTON WISHART, Toronto, Canada: Many references are given which support the view that infection in the mastoid antrum is the cause of acute intestinal intoxication in infants.

The medical staff of the Hospital for Sick Children, Toronto, believe that the type of the disease seen in Toronto is essentially the same as that which exists in other parts of Canada and the United States. Study of the disease has been pursued seriously for five years. For the last two years a large body of workers have co-operated.

The facts and figures used are drawn from an elaborate study of all pertinent records for five consecutive years—1925 to 1929, inclusive—at the Hospital for Sick Children, Toronto. The statistics and conclusions have the consideration and approval of the Physician-in-Chief, and his research department and the pathologist and his department. The relationship of infection of the paranasal sinuses is not considered in this report.

The onset of the disease is rarely characterized by a "cold." The great majority of the infants were without any clinical evidence of an upper respiratory infection at the time they were toxic. Many infants remained without any ear infection throughout the whole course of the illness. Both ear drums of many showed abnormality immediately preceding death. This change is an antemortem phenomenon due to forcible ejection up the eustachian tubes. When mastoid infection exists it is the result and not the cause of the child's lowered condition.

Accumulations or infections found in the mastoid antra of infants at autopsy are antemortem in origin.

Mastoid antrum puncture for diagnosis of latent mastoiditis is not to be recommended. Bilateral mastoid operation as a cure for the disease was a failure. Operation is to be postponed as long as possible. The autopsies of two intensively studied series of cases of acute intestinal intoxication show that mastoid infection was not common.

There was no correspondence between the bacteriology of the infection in the upper respiratory tract and that in the intestinal

tract. Evidence is being accumulated to show that the disease is of intestinal origin.

Conclusion: Infection of the mastoid antrum is not the cause of acute intestinal intoxication in infants.

DISCUSSION.

Dr. R. T. Atkins, New York: Dr. Wishart deserves a great deal of credit for his painstaking investigation. He apparently began his study with an unbiased mind, and as his conclusions are at variance with the opinions of a number of investigators, his communication is an important one and deserves free discussion.

A paper which is so full of statistics is difficult to discuss, and as I have very few statistics to present, I can only give my impressions, which have been gained by observation of these cases, in conjunction with the pediatric service at Bellevue Hospital.

Infants with acute intestinal intoxication or acute enteritis have been admitted to the pediatric wards of Bellevue Hospital, with no evidence of infection of the ears, noses or throats. It is possible that an infection may have preceded the enteritis, but at the time of admission there was no evidence, either from the history or examination.

Many infants developed otitis media in the wards and were treated by myringotomy, antrotomy, or more extensive mastoid operations, as the condition warranted. Many of these infants died; a few recovered.

Acute enteritis associated with lower respiratory infections has also been observed. The pediatricians at Bellevue believe that an acute infection anywhere in the body may precipitate an acute enteritis.

On looking over the records of infants under two years of age, who were operated on for mastoiditis during the past few years, I was only able to find histories of two infants who were transferred to the pediatric service because of enteritis. I have never seen infants with this condition in private practice, nor have I seen cases at the New York Eye and Ear Infirmary. Apparently it is primarily a pediatric problem, particularly among the undernourished hospital babies. These infants develop otitis media very easily because of their low vitality, relatively short and open

eustachian tubes, and the presence of embryonic tissue in the middle ears.

I believe that the ear as an etiologic factor in enteritis in infants has been greatly over estimated.

Dr. Charles McKahnn, Boston: If the otologist is in difficulties in regard to this controversial subject, what of the pediatrician?

At the Infants' Hospital of Boston otitis media with mastoiditis is found in 50 to 60 per cent of the autopsies made on infants dying from acute nutritional disturbances. The lesions, however, are recent and microscopically show little infiltration of the walls of the mastoid. Also otitis media has been found in almost 50 per cent of the autopsies of infants dying from other causes. We feel that these findings are quite representative because in our hospital about 80 per cent of the babies that die come to autopsy and the series of nutritional cases is entirely comparable to the non-nutritional cases. Otitis media of the type found seems to be so common as to represent a manifestation, probably terminal, of relatively very little importance.

We admit and recognize fully that diarrhea may occur with almost any parenteral infection. But the incidence of diarrheal disease does not appear particularly high with any one type of infection, nor are diarrheas due to parenteral infection as a rule so severe as those of acute intestinal intoxication. Diarrhea does not occur often in patients ill with acute otitis media. Acute intestinal intoxication as it occurs in great outbreaks is a summer disease, and also a disease of the poorer classes, with much lower incidence in private practice than in charitable hospitals. Otitis media, on the contrary, does not show either this seasonal variation or the diminished incidence in private practice.

The results of operation on the mastoid in relieving acute nutritional disorders you will have to determine. The reports are at variance and I should not care to criticize operative results before the men who have done the operations.

A theoretical consideration is worthy of note. I believe it would require a very powerful toxin, produced in such a small place as the infantile mastoid, to produce such a severe intoxication as is found in acute nutritional disturbances. No common

organism has been shown to have a toxin powerful enough to produce such results. Comparative mortality statistics in acute nutritional disturbance are of interest.

Dr. Marriott reports in one series of cases a mortality of 35 per cent in patients treated by mastoid operation. Our own mortality has averaged about 35 per cent. This figure seems to have been usual in other places also. It is near the figures reported by Powers from New Haven, and Schloss from New York, for a series of cases treated symptomatically, and without mastoid operation. In the summer of 1927, Dr. Richards operated on many of our cases at the Infants' Hospital. In the same summer at the Boston Floating Hospital, a similar series of cases was treated without operation. The case fatality from acute nutritional disease was almost identical in the two hospitals.

From our observations at the Infants' Hospital of Boston, we are inclined to believe that although otitis media may be responsible for some nutritional disorders, as may any parenteral infection, the extensive and severe condition which is usual in cases of acute intestinal intoxication is not due to otitis media or mastoiditis. Rather, in the great outbreak of acute nutritional disease, otitis media is a terminal manifestation, developing within a short time of death.

Dr. Robert Sonnenschein, Chicago: Much has been said about the middle west. Chicago is less than 300 miles from St. Louis so that it may almost be considered a suburb (!) of that town. I would like to say a few words which will be a repetition of what I said before this society three years ago. I believe that some of the work that was done in St. Louis has been misunderstood. Shortly after a certain meeting in St. Louis, the pediatric staff of the Michael Reese Hospital became enthusiastic about these operations, and against our better judgment, many of these cases of infants from six weeks to six months old were operated upon because the pediatricians insisted upon it. None of these cases recovered. We could not see any marked changes in the drum membrane. In small infants it is difficult to note whether there is a bulging of the drum membrane at the superior angle of the canal or not. In a few cases of athrepsia infants died who had not been operated on. There was fluid in the an-

trum, perhaps due to infection of the tube or the passage upward of stomach contents, etc. Since then I have not operated any of these cases, but have operated only those which showed otologic pathology, and which would ordinarily be operated upon whether gastrointestinal infection was present or not.

Dr. Arthur M. Alden, St. Louis: From the text of this paper and the general sentiment expressed in the discussion of it, it might appear to some of you that I have taken a rather first class lambasting this afternoon. However, I am quite sure that the last word upon this subject has not as yet been said. Dr. Wishart's paper is an important and timely contribution and a definite step towards the clarification of this subject about which there has been so much discussion and so much misunderstanding. Just why in the East, competent observers should have had one type of result, and why in the West we have seen different results, makes one wonder. I think the answer lies largely in the fact that we are not all talking about the same thing. I am sure that the pediatricians we have heard this afternoon are discussing something entirely different in the way of gastroenteritis from those cases which we have previously referred to as due to ear disease.

This work was begun in the autumn of 1924, and upon my shoulders fell most of the otologic end of this investigation. That Fall we saw a great many babies suffering from advanced anhydremia, due to vomiting and diarrhea, where the pediatric service had ruled out all ordinary causes of these conditions, but where careful ear examination demonstrated definite otitis media. Of these cases seventeen came to operation. All these babies showed the typical symptoms of middle ear infection and many of them had had repeated myringotomies. No case was operated except upon definite otologic indications of mastoiditis, the most common symptom being a distinct sag of the posterior canal wall, which I feel is pathognomonic of an infection in the mastoid antrum of the infant. I would like to say here that this sag in the posterior wall must be carefully differentiated from the downward bulge of the drum membrane which occurs in the presence of an attic infection in the infant, and which is due to the peculiar angle which the tympanic membrane in a baby

makes with the superior canal wall in whom it is much more obtuse than in the adult. Of these seventeen babies which came to operation, we were able to save nine. All of them were desperately ill and many of them were considered almost moribund by the pediatric department.

The following summer I remained in St. Louis for the sole purpose of carrying out a careful examination of the ears of those babies which we always see at that time of year with the various forms of summer diarrhea. Every case of diarrhea and vomiting was examined time after time, but in no case that summer did I find any indications for otologic intervention. The answer to this, of course, is that the syndrome occurring in the summer was due to some cause other than in the cases that we had seen a few months before. The next Fall, beginning in October, we saw more cases where we felt that the intestinal upset was due to ear infection. Dr. Lyman was in charge at that time, and because we had learned what to look for in these cases and to diagnose them much earlier, his operative results were better than mine. The following year a much smaller number of cases was observed, and this last winter, although I have seen many babies with otitis media, there were only two who showed gastrointestinal manifestations which were definitely due to the ear infection. In each of these the operative procedure yielded results that were little less than spectacular.

There is no doubt in my mind that there is a type of infection or a type of baby where an otitis media may be directly responsible for gastrointestinal symptoms that can be profound enough to produce anhydremia and death, and if this type of case is treated according to the indications which we have laid down, satisfactory results will be obtained in the majority of instances. We have never said that ear infections were the sole agents responsible for gastrointestinal disease in babies, but we do say that otitis media may be responsible for this condition in certain cases.

None of our babies were operated upon without definite otologic indications and the results obtained in the gastrointestinal syndrome were only incidental. We do believe that in many

of these cases, the ear disease is overlooked, because careful daily ear examinations are neglected, and in those cases where a diagnosis of mastoiditis was made we merely gave these babies the benefit of the same type and standard of treatment that older patients have been receiving at the hands of our best otologists for years. The changes in the texture of the ear drum in babies with profound anhydremia, but without otitis media, to which Dr. Wishart refers, we have observed many times; but like him, we have felt that these were merely local manifestations of the general condition and not due to infection in the ears.

The question was raised as to whether most of our cases occurred in undernourished clinic babies. To this I can say definitely no. Some of my most spectacular cases came from homes in which they had received everything that wealth and intelligence could give them. I remember one child in particular, a girl of eight months, whom I saw about three years ago. During the two days before I saw her, she had had daily fifteen to twenty watery stools and much vomiting. This condition had begun following an acute cold. The first time that I examined this child, was in the morning, and I could find nothing in the ears upon which to make a diagnosis of infection. However, when I saw this baby again in the evening, both ear drums were gray and lusterless and there was some reddening around the periphery and along the handle of the malleus on each side. The child was very sick with high fever and a leucocytosis of 26,000. She was sent to the Children's Hospital where I examined her again the next morning and incised both drum membranes which were now bulging in the lower half. Before night the diarrhea, vomiting and temperature were all much improved, and three days later she was discharged from the hospital apparently entirely recovered. Two days after she returned home the entire gastrointestinal picture returned and she was brought back to the hospital. Examination at that time showed the left ear to be normal, but on the right side the drum membrane was bulging and there was a distinct sag on the posterior wall. Myringotomy was done and the child was treated by the pediatrician for several days with no relief. Because the ear condi-

tion persisted, I did a mastoid antrotomy on the one side and within twenty-four hours all of her gastrointestinal symptoms had stopped. Following this she made a complete recovery. During the next year I saw this child on several occasions in consultation with her pediatrician. With almost every cold she would have some diarrhea and often pain in the right ear, that is the one which was not operated previously. Myringotomy on several occasions produced a complete cure. At the end of about a year she developed a very severe attack in which incision of the ear drum failed to produce relief from her diarrhea. This time she was readmitted to the hospital, the right ear was operated and a pure culture of hemolytic streptococcus obtained from the mastoid antrum. This baby has been well now for more than three years.

This piece of work, like all new things, was received with skepticism, and later, particularly on the part of pediatricians, with an unwarranted enthusiasm. As a result, many needless mastoid antrotomies have been performed and many babies have probably succumbed, due more to the operation than the condition for which it was done. In many of the reported cases the bacteriologic work was poorly done or not done at all. What is the answer to the whole problem? My own conception thereof is that it is either the reaction of a particular type of baby to an ear infection, or the reaction of a baby to the specific type of infection with some strain of the hemolytic streptococcus. Probably the latter is more near to the truth because in our experience, these infections have occurred in waves like an epidemic. From an otologic standpoint, there is nothing new in this conception except that a baby may have an ear infection severe enough to produce gastrointestinal symptoms which will rapidly dehydrate and kill him, but may show neither external signs of mastoiditis nor a bulging ear drum. Careful ear examination will, however, demonstrate the other signs that are pathognomonic of mastoid infection. If you look for these cases carefully enough, you will occasionally see them in your own practice.

Dr. G. M. Coates, Philadelphia: This whole subject has been highly controversial for some years past, and this very careful study of Dr. Wishart has possibly made it more so than ever,

with the result that we shall have to decide before very long in which camp we are going to be. It is difficult to reconcile reports of recovery of infants after middle ear and mastoid antrum drainage with the conclusions of Dr. Wishart, who states that mastoid drainage has had little or no influence on the intestinal condition. I have talked this matter over with the pediatricians in Philadelphia during the past two or three years, and from their point of view, mastoid infection does not play a large part in the production of gastrointestinal disorders. One or two prominent men have told me that they thought it did have some such influence, but on the other hand one of our ablest pediatricians says that he has not seen these cases, although looked for very carefully. In a number of cases in the pediatric department of the Graduate Hospital, the otologic department was called in, and a number of mastoids were operated upon but never without some definite otologic reasons. The results have been disappointing, as regards saving the lives of the patients, although I have seen cases in which a simple myringotomy or an antrum drainage played a large part in the recovery of the patient who was rapidly going down hill, who after this procedure promptly turned toward the road to recovery. These cases in the Middle West are reported largely as cases of middle ear infection, which view does not seem to be confirmed by Eastern observers. Not so very many years ago (about the 70's) it was found that children dying with intestinal disease showed evidence of mastoid infection. This was attributed to secondary infection from injection of stomach contents through the eustachian tube, and also to the lowered resistance of the patient, due to the intestinal condition. I have been on the fence in this matter. I think there are cases where elimination of the mastoid infection allows the child to get well from the intestinal disease, but we do not see many such cases in Philadelphia. If both these views are true I believe that we are talking about two different conditions.

Dr. Lyman Richards, Boston: About three years ago at a section meeting of the American Medical Association in Dallas, I reported with some timidity on a few unsuccessful results of operations on infants with acute nutritional disease. At that time

Dr. Lyman of St. Louis assured me that I would return home and pursue the work, good results would follow. I regret that this has not been the case. I recently published in Archives of Surgery, where (otologically speaking) it is buried in the Cushing Birthday Supplement, an account of one hundred cases of acute nutritional disease. Among these were twelve single or double mastoidectomies, of whom nine died, figures quite in accord with Dr. Wishart's results. So far as I have reviewed them the results of operators west of the Mississippi have been better and more encouraging than those in the East. Why is this?

Such intersectional differences in results obtained by competent men using the same technic can scarcely be due to anything connected with the operation itself. The burden of proof, of course, rests with the man who fails to cure the patient, and no one doubts the accuracy of the Western reports of success. In the East we have found much the same pathologic conditions, both before operation, at the time of operation, and alas, at the postmortem. Our divergence has been in the operative result. I am forced to conclude that in spite of the similarity in the written descriptions of these cases, we in the East are not dealing with the same clinical entity, otherwise our results would have been better. Description of cases in which it was decided to operate two weeks after the case was admitted rarely applied in my experience, since the disease was rapidly fatal, usually within a week. Incision of drum membranes frankly or suspiciously pathologic, usually had no effect on the fever; likewise, the opening of a mastoid, mildly or violently involved, did not avert a fatal outcome. The patients seemed so toxic and desperately ill that even if they had a mastoid infection (as I am sure they often did have), drainage of it did not avail. I have come thus to regard such an infection as a secondary complication in a greatly debilitated patient, not as the primary cause. Almost never were any otologic symptoms noted at the time of admission. They developed later, as any complication does. Moreover, why is it that during the treatment of hundreds of mastoid infections, of all grades of severity, this picture of nutritional disturbance almost never occurs?

In conclusion, let me say that my experiences have been wholly in accord with those of Dr. Wishart. I feel that we will never come to a complete understanding of the problem until we institute the custom of exchange surgery. Let an Eastern man go West and actually observe the type of patient that can be cured by operation. Let the Western man come East during one of our epidemics and operate on some of our cases. Personally I extend a most cordial invitation to Dr. Dean or Dr. Alden or Dr. Lyman to come and stay as my guest and remain as long as he wishes, if he will demonstrate that my type of patient can be cured by operation.

DR. WISHART, Toronto (closing): To some of the gentlemen who have discussed my paper so kindly, I have nothing to say but—thank you very much. You have been very complimentary and you seem to be quite in accord with my views, but there are one or two points which require mention.

I could not read my whole paper, but simply summarized some sections and omitted others. Apparently in so doing I gave one false impression. The majority of the infants of this report were not undernourished. There was a high percentage of well nourished babies.

Dr. Coates states he is still uncertain. He wonders whether the recoveries reported by others were due to operation or to some other factor. This makes me refer to another point. In the two series of 1928 and 1929, we sat in conference until we came to unanimous conclusions. One of these was that in every case but one there was an explanation for the cause of death other than the ear or the mastoid. We had one patient die who did not come to autopsy, in whom there was no other known source of infection except that found in the ear. In most cases the drum membrane remained normal until just before death and then took on a pathologic appearance. In not one such instance did recovery take place if incision of the drum membrane was made at that stage.

Dr. Alden should be assured that no lambasting was intended. We pursued our studies without any bias one way or the other. He asks me how the ear was examined. An otoscope was used invariably—but sometimes a head mirror was also used. I am

very glad that he agrees with the final appearance of the drum membrane as part of the general anhydermic picture. I feel that Dr. Alden's remarks support my paper. He admits that cases in which ear intervention was indicated or would have altered the course of the disease were rare. Such a statement supports my conclusion that the ear infection is not the main cause of the disease.

From all this discussion has emerged one point of great importance to the otologist. In many pediatric articles the extreme view has appeared suggesting that since the cause of acute intestinal intoxication was in the mastoid, otologists must operate at the behest of the pediatrician. Dr. McKahnn showed that this view is no longer held in many of the larger centers, and this is a great change. Otologists should operate only when they are sure that the condition will be benefited by the operation and not otherwise.

MORNING SESSION, WEDNESDAY, MAY 21, 1930.

The Clinical Problems of Chronic Suppurative Otitis Media.

GEORGE E. SHAMBAUGH, M. D., Chicago: It is apparent from the multiplicity of symptoms which present themselves in cases of chronic suppurative otitis media that no one form of treatment is likely to be suitable for all cases. First of all comes the decision as to whether an ear should be subjected to conservative local treatment or whether the situation calls for radical surgical measures. The time has long since passed when every chronic discharging ear is looked upon as a possible menace from a serious complication. Only those cases where such a menace exists are properly subjected to radical surgical measures. The differentiation of the dangerous from the nondangerous type of running ear rests upon the type of local process which involves the middle ear, whether this is a disease which involves the middle ear, whether this is a disease which involves only the mucosa or whether there is an actual bone invading process, caries or cholesteatoma. It is only in the latter that radical operative measures are to be considered. The operation is aimed at eradicating this focus, which is the menace of a possible complication. The operation is not undertaken primarily to cure a chronic running ear of its discharge. Where the radical operation has been carried

out, and the cholesteatoma or caries removed from the attic, the aditus and the antrum, the operation has been a success, even though some moisture may persist from the tympanum or tube.

A discussion of the indications for the line of treatment to follow consists of a discussion of the recognition of the pathologic changes which are associated with chronic discharge. Not all cases of caries or even cholesteatoma are such that radical surgery should be advised, and not all cases where local, conservative treatment fails to check the discharge are in need of a radical operation. On the other hand, no form of conservative local treatment can relieve a patient of the menace of a serious complication in those cases where there is an indication for radical surgery.

Conservative Treatment of Chronic Suppurative Otitis Media.

DR. GEORGE L. TOBEY, Boston: In the treatment of a chronic suppuration one of the most important points is to convince the patient that treatment really is very necessary. The indifference with which a patient usually regards this serious condition is remarkable, with the result that many lives are lost annually which might have been saved had the sufferers been aware of the dangerous nature of the disease. In this treatment of the suppuration of the middle ear there are certain principles to be served in all methods. These points include an active treatment, and an accurate history is essential; the presence and duration of intermittent discharge, nasopharyngeal infections and their effect on the aural condition, local pains, headaches, vertigo, variations in hearing, tinnitus, etc., should be ascertained. Intermittent otorrhea is as a rule due to recurring infections of the eustachian tube, and is confined in this section to the mucous membrane; a constant purulent discharge is due to the presence of a bone necrosis and cholesteatoma. The otologist's first duty is to correct pathologic changes in the upper respiratory tract. Irregularities of the nasal septum are seldom a factor in aural infection. Hypertrophic and suppurative infections of the sinuses—the former a source of recurring acute rhinitis, the latter a constant source of focal and direct infections, must be corrected by necessary operative procedure. Chronic empyema of the antrum is often overlooked. Infection of the posterior ethmoid cells and of the sphenoid sinus must be considered. Adenoids are often a

factor in recurring chronic infection of the eustachian tubes. The tonsils should be removed when proven to be either a pathologic or a mechanical factor.

The treatment of suppurative otitis itself is classified under three general headings, depending upon the lesion and extent of the pathologic process: (1) Local therapy: (2) intratympanic operation; (3) radical surgical interference. Under the first heading the usual local measures of cleansing, the careful removal of polyps and the use of alcohol, etc., are indicated. Ionization has been used, especially by our English confreres, and may be considered as a valuable adjunct to treatment in many cases. In the absence of intracranial complications, and where the X-ray demonstrates that the area involved is limited to the middle ear, annulus, attic and antrum, the essayist proceeds with an intratympanic operation, bearing in mind and advising the patient that should this relatively conservative operation prove insufficient, a radical mastoid may be performed later. The operation of ossiculectomy is indicated when a purulent inflammatory process of the middle ear does not respond to local therapy as outlined, and in cases where the diseased process is chiefly confined to the membrana tympani, the ossicles, the antrum and the tympanic walls.

The complete radical operative procedure is indicated: (1) "When a permanent cessation of the purulent process has not been affected by prolonged local intratympanic treatment, combined if necessary with such other operations as removal of granulations, enlarging perforations, etc. (2) When a cure has not been effected by the removal of the necrosed ossicles and the curettage of the middle ear. (3) When acute symptoms of mastoiditis supervene in otitis media purulenta chronica. (4) On the appearance of chills, fever, vertigo, pain or other unusual symptoms. (5) The appearance of facial paralysis during the course of chronic purulent otitis media. (6) Attacks of vertigo, nausea and vomiting, indicating that the necrotic process involves the labyrinth. (7) In all cases of complicating intracranial or lateral sinus involvement. (8) When there are positive symptoms of cholesteatoma in the mastoid. (9) When there are fistulous openings in the cortex of the mastoid process or in the osseous canal

wall. (10) Whenever extreme depression or other symptoms of disturbed mentality accompany the disease." (Phillips.)

As Siebermann well said: "We must not only know how to employ conservative treatment, but we must have a clear guiding principle as to how long we may do so.'

The Radical Mastoid Operation.

Dr. J. Morrisey Smith, New York: The object of the radical operation is the conversion of the external auditory canal, the middle ear, attic, antrum and mastoid into one continuous open cavity. In order to accomplish this, the mastoid antrum is opened, the posterior bony canal wall which separates the middle ear from the mastoid is removed together with the malleus, incus and drum membrane; then the diseased granulations are removed, converting these spaces into one cavity. The postauricular incision is completely closed with sutures, and the after treatment is carried out through an enlarged membranous external auditory meatus. The radical cavity is cleansed and cared for daily until it becomes dry and completely dermatized.

The intimate relation of the middle ear and mastoid with the facial nerve, internal ear, jugular bulb and other intracranial structures makes it absolutely necessary for the operator to have a practical knowledge of the surgical anatomy involved in this operation. Its success will then depend upon a very careful and exacting performance of the different steps comprising its technic.

The steps of the radical mastoid operation:

The mastoid incision and exposure of the cortex.

Entrance to the mastoid antrum.

Removal of the posterior bony canal wall.

Removal of débris from the middle ear.

Preliminary lowering of the facial ridge.

Removal of the external attic wall.

Removal of the annulus tympanicus.

Curettage of the eustachian tube.

Final reduction of the facial ridge and exenteration of the hypotympanic space.

Lowering of the floor and anterior canal wall.

Inspection of the radical cavity.
Cutting the flap.
Closure of the wound.

Close attention to all details is necessary in order to insure complete removal of all diseased bone, leaving a large, smooth cavity which can epidermize later. Great care must be exercised in reducing the facial ridge so as not to cause injury to the facial nerve. In curetting the orifice of the eustachian tube one must avoid fracturing the inner wall of the tube and injuring the internal carotid artery, and a word of caution is also necessary regarding the removal of the tensor tympani muscle and the processus cochleariformis. It will be noted that the base of the ligament which arises from the tensor tympani muscle is attached to the internal wall of the inner ear just below and slightly posterior to the point where the facial nerve starts its horizontal course backward through the fallopian canal. Attempting to curette the muscle at its termination or the ligament at its base is very apt to result in injury to the facial nerve.

DISCUSSION.

Dr. Alfred Lewy, Chicago: In the forty-one years of the existence of the radical mastoid operation many changes have been made in the plastic, and the bone work has been modified toward conservatism or toward wider exposure, as the case demanded, by Jansen, Heath, Blackwell, Bondy, and we now add Dr. Smith's name. Its indications and contra-indications have been more definitely established, and in many instances it becomes a life saving procedure, but it has not yet given us definite control over the healing process, and so far as restoration of function is concerned, that is not to be expected. Even though the ear has been made "safe," persistent discharge is common. Under an epidermized surface inflammatory changes and cholesteatoma may progress in the bone, as shown by Neumann.

Lange, Thies, Von Eicken and Hinsberg favor the operation via the canal, often under local anesthesia in sclerosed mastoids, while Neumann and Loebell believe the approach via the mastoid cortex better and safer, except in carefully selected cases. Kuemmel, a former adherent of the canal route, after a fatal case of sepsis

attributed to this technic, has returned to what he calls the "only seemingly more elaborate approach behind the ear." His case started with infection in the plastic flap. My own practice is to do the Stacke only when a forward lying sinus makes it the operation of choice. Dr. Smith does a most meticulous operation, but in case of an intracranial complication or labyrinth exenteration, when time is a consideration, it may not be advisable. In fact, ordinarily I remove only gross pathology from the tympanic cavity, curetting as little as possible. I am skeptical of the value of tubal curettage except in the presence of manifest pathology in that region, and, if possible, I like to preserve the anterior part of the membrana tympani to try to cover the tubal ostium. I also leave the stapes region alone if there is any vestibular function or hearing, but I do try to remove carefully the overhanging facial spur, as described by Dr. Smith, and for cutting down the bridge prefer the flat chisel. A properly sloped cavity is desirable, but I have not tried removing so much of the tip. In general, I follow Neumann's rule: "The simple mastoid as radical, the radical as conservative as possible." The type of operation and the form of flap should be selected according to indications. In a central perforation with chronic discharge, if operation has to be done, a simple antrotomy may suffice. Dr. Tobey's ossiculectomy seems to be virtually the Stacke operation without the posterior incision, and done by special forceps instead of by Stacke or Thies chisels, which constitutes that much improvement.

Dr. Shambaugh's indications and common sense views, based on a broad experience, appeal to me strongly. They should supplant the usual textbook teachings. The ordinarily accepted urgent indication for a radical is the threat of an intracranial complication. On the other hand, a labyrinthitis may be a contraindication to any surgery at that particular time. Other indications may be classed as relative. One accepted as important is cholesteatoma with perforation in the membrana flaccida. On the other hand Scheibe reports that he has operated only 3 per cent of his cholesteatoma cases with no deaths among either the operated or conservatively treated groups. The patients were personally and meticulously treated. Frey also takes a very conservative stand. This leads us to what I consider one of

the most important considerations, viz., the patient's ability and willingness to take persistent treatment, and the doctor's willingness to give careful personal attention to the case. Under quantity production methods, or in rural or proletariat communities, or with an unintelligent clientele, ultraconservative methods are not practical. Another condition frequently stated as indication for immediate radical operation is an acute exacerbation of a chronic suppuration. I prefer hospitalization and awaiting subsidence of the acute symptoms, but operating immediately if the course is unfavorable, and have had so far no occasion to regret this procedure. With an intelligent clientele, economically situated so as to take treatments over a period of time, very few radical mastoid operations should be necessary.

Both in the conservative treatment and the after treatment of operated cases we are dependent upon the activity of the tissue cells and the body fluids. We are beginning to learn the value of bodily vigor in terms of endocrine efficiency and vitamin supply. Perhaps it is not too much to hope that in the investigations fostered by the Society into the underlying causes of otosclerosis other than heredity, some so far unclassified factors may be discovered underlying chronic suppuration and other chronic conditions. The laboratory is beginning to reveal the local action of the macrophages in tissues cultivated outside the body, and the activating and antitoxic properties of the body fluids. This may seem academic, but as practical suggestions I would mention heliotherapy in sluggish wounds; exercise, diet and cod liver oil in lowered immunity. A study of the effect of heat and cold on inflammation and repair would be desirable; their effect on the macrophages and on hyperemia, and whether or not the hot compress, the ice bag and the hot flat iron are not practically as good therapeutically as the much vaunted and expensive physiotherapy apparatus so much in vogue.

In stubborn tubal suppurations after radical mastoids I have had some success with a treatment recommended by Voss, viz., suprarenal extract followed by a drying powder, in my case boric. This has succeeded in one case after failure of zinc ionization. I do not know anything better than Dr. Tobey's conservative treatment technic, and the nasopharyngeal treatment

is eminently fitted to tubotympanic cases. My usual routine is the well established attic irrigation (even in central perforations) with 50 per cent alcohol, followed by boric powder. Perhaps the iodin boric powder or other preparations may prove more useful.

As to the plastic, Bondy now merely slits the canal wall the depth of the antrum; Bárány does no plastic at all with apparently fair results. My own observation of a few cases is favorable, but they all presented small cavities. Some of Bárány's failures were cases in which a pocket formed behind the canal wall. It is quite evident that the plastic should be made to fit the case in hand. One precaution is that the flap shall not cover exposed dura. In one case in which I did a Stacke, I afterward thought it would have been wiser to sacrifice entirely a tongue flap in order to supervise better the operated area. With skin grafts and spiral flap in the tegmen-facial ridge space I have had no experience.

Dr. Edmund P. Fowler, New York: The prime requisite (whether the suppuration is chronic or not, and whether we do a radical or a simple operation) is efficient drainage and efficient protection against subsequent infection. As regards treatment of the tube, it is more necessary to treat the nose and nasal sinuses from which the tubal reinfections are regularly acquired, than to treat the tube itself, and the middle ear with its spaces and connecting cavities is an extension of the nasal sinuses. I have found that in 86 per cent of diseases of the ear in children, there is an involvement of the nasal sinuses. For inspecting these cases I use a little instrument which magnifies about four times. I use it also in intratympanic operations. It consists of a pair of telescopes mounted as spectacles. One may look above or below it. Above each telescope is a light connected with a separate battery. This brings each light in the axis of its telescope, and enables one to obtain a light without any shadows. With this apparatus the stapes looks enormous and there is no difficulty in dissecting around it during radical operations. It is surprising to note how many cells are still present and require curettage when one thus inspects the wound after a radical.

DR. G. L. RICHARDS, Fall River, Mass.: I would like the advice of Sir Charles Ballance as to how we should treat the small children who crowd our clinics suffering from suppurative otitis. Shall we do radical operations or shall we try to handle them as Dr. Shambaugh has suggested. We cannot hospitalize them all. Yet they do not get good home treatment to follow up the treatment in the clinic. I find the only thing to do is to clean them as well as we can and then have the mother use the alcohol boric acid drops, showing her how they should be used, after wiping the ear dry. In spite of all we can do, many of these children are going to be deaf and an economic problem. I do not believe we accomplish any more for these cases than we did thirty years ago, except that the removal of adenoids and tonsils has become more general and this helps the ear condition. I have had some good results from the iodine powder. One case who was to have had a radical operation was given the iodine powder pending the selection of a date for the operation, and to my great surprise the ear became dry and has so remained. Too short a time has elapsed to say whether this dryness is permanent.

DR. FRANK R. SPENCER, Boulder, Colo.: We have used zinc ionization for some of our patients. It has improved most of them, but is not ideal. I am sorry more has not been said about conservative operations. Dr. Smith showed some on the screen and Dr. Tobey referred to ossiculectomy. I like to see the operative field better than can be done during an ossiculectomy, and prefer some type of conservative radical operation. I began my professional career the same time Dr. Tobey did, and just when ossiculectomy was passing out. In regard to dense bone, the electric burr has helped me a great deal. I often use it in the simple mastoid operation. I believe a great deal of radical mastoid surgery can be avoided by more conservative procedures.

DR. JOHN F. BARNHILL, Indianapolis: Members will recall that about five years ago a paper was presented before this Association entitled "One Thousand Cases of Chronic Otitis Media Cured Without Operation." At that time some who discussed the paper accepted the view of the author that operative

interference was seldom indicated in the treatment of this affection. Other speakers, including myself, agreed that while cure of chronic suppurating ears by local applications is possible in a considerable percentage of cases, there are without doubt many others in which the pathology present is such that cure is not probable except through operative means. It was, therefore, the opinion of the latter that both the paper and the discussions led to unwarranted optimism from medications, and that to follow such course would encourage delay that might lead to danger which should be anticipated and more rationally met.

The symposium we have just heard is, I believe, based upon more tenable grounds. The first paper states clearly the pathology which justifies local treatment in one case and operative interference in another. This is as it should be, for every clinician knows that cases differ widely in these respects.

I wish always to be conservative in things surgical, but feel that conservatism embraces operative as well as therapeutic measures in the treatment of chronic otitis media. Methods of treatment must always be determined by the nature of the pathology which is present. If the pathology is limited and accessible, conservatism consists in local treatment; but if extensive, deeply seated and inaccessible to local medication, then true conservatism is exercised only by employment of surgical measures. This view has been well expressed by the first essayist. The writer however used one expression, which (if I understood it correctly) I believe should be eliminated. The statement was made that pus may be present in the ear without harm to the patient. Suppuration of the middle ear constitutes a focal infection, and, unless many able observers have been mistaken, focal infection is the primary source of many local and general systemic diseases, some of which are serious and even fatal. It is, therefore, a well founded view that such a focus should be eradicated by local treatment if possible, but by radical surgery if necessary.

Ossiculectomy as an efficient measure in the treatment of certain types of chronic otitis media has again been revived. This operation was used, as Dr. Tobey stated, more than twenty-five years ago, and then was superseded by other means. At the period when it was in vogue I performed many ossiculec-

tomies, and can testify to the value of the procedure since many cases were relieved of their discharge. As I look back upon this plan, however, and compare the steps of the performance with those of the more recent modified radical mastoid operations, I am of the decided opinion that the latter operation is safer, more easily performed, conserves hearing better, and is more certain of satisfactory results. I am not convinced, therefore, that a revival of ossiculectomy would be a step forward in the treatment of chronic running ears.

Dr. Joseph H. Bryan, Washington, D. C.: I do not know whether Dr. Smith was responsible for this symposium or not. I had a conversation with him at Atlantic City and the subject of chronic otitis media was brought about as a result of a paper I read at Washington. I said chronic suppuration was a menace. Dr. Shambaugh thought I overemphasized the fact. I believe it should be emphasized on account of the attitude of the general physician.

Dr. James A. Babbitt, Philadelphia: I have wondered if there was not a course between the conservative method employed by Dr. Shambaugh and the somewhat radical procedure with ossiculectomy and opening the mastoid wall, by Dr. Tobey. In the outer portion of the attic cavity, we encountered many obstacles to free drainage, often reducing the channel to a very narrow tortuous canal. I have attempted in the past two years to enlarge this channel by simply probing beneath the outer wall of the attic with blunt probes and a simple attic curette. When perforations were present in the Shrapnell area an opening was made in the lower posterior quadrant. Lower anterior perforations and marginal perforations were enlarged into an opening in this position, in order to always maintain drainage directly downward. In my list of ten cases one, a child, failed, but nine ears have remained practically dry; one case in an influenza attack flared up for a few days but promptly subsided. It seems to me that this very simple method will show considerable value.

Dr. Jacques Holinger, Chicago: With reference to the treatment of chronic suppuration, it is important to watch the eustachian tube, the nasal end, as well as the middle ear end. I never forget to inflate the middle ear and control the result by means

of the hearing tube. A question of physiologic importance is this: Why do we get improvement of hearing in many cases after inflation when there is a large perforation of the drum head? I was the first one to do the radical mastoid operation on this side of the Atlantic. In the spring of 1893 I did six or seven at the Eye and Ear Infirmary in Chicago, and in 1899 showed several cases cured after six years. Nobody wanted to follow that technic. My friends said, "What is the use of spending so much time?" Concerning the indications, I do not believe there are many fundamental changes since then. If you look up the first number of the Berliner klinische Wochenschrift for 1893 you will find that you cannot add very much to it. Many modifications that were suggested were due to individual anatomic or pathologic conditions. They will be suggested to any operator, who is guided by the findings. One thing I am surprised at, and that is the ever recurring question of suppuration and nonsuppuration of the radically operated cavity. The latter is lined by a scar which forms scales and these must be removed from time to time, or they become macerated and suppurate. That is in the nature of things. Scar tissue is not normal tissue; it must be considered as pathologic tissue and proper precautions taken. Those of the gentlemen who have experience in general practice will remember scars from old ulcers of the lower limb, or scars from large osteomyelitic wounds. They are all poorly nourished epidermal scars. The analogy is evident. Some do very well and give no trouble. Others can hardly ever be made clean and dry.

Dr. George E. Shambaugh, Chicago, closing: I hardly know what to say in rebuttal, as I think that everything I can say has already been stated in the paper. The old idea that every discharging ear is a menace from which a complication may arise has long been discarded. It is only the exceptional case of chronic discharging ear that constitutes the menace of a serious complication. The usual situation is one of a mild tubotympanic process. Dr. Bryan asks how we are going to differentiate between these two types. My paper was a discussion of just this point and if I have not made it clear in the paper I certainly cannot do it now.

Dr. Ballance harks back to a solution of this problem which I thought had long since been discarded, namely, that if a discharging ear cannot be cured in a few weeks' or a few months' treatment, the patient should be advised to have a radical operation; and he quotes a case of a patient suffering from suppurative ears for thirty-five years, being under the treatment of numerous physicians during this period who had not been able to cure the ear trouble, and finally the patient succumbed to an intracranial complication. That is not an unusual situation. It is apparent that one of two things was at fault. Either the men who had undertaken to treat this patient's ears did not recognize that this belonged to the class of cases where there is a bone invading process and so did not warn the patient that a complication might arise, or what is more probable, the patient had been warned often enough that the ear situation was a menace, but did not care to consider having a radical operation.

I suspect that we are, all of us, not so far apart in our views as the discussion might lead one to believe. I do not believe that any otologist today advises operation on a running ear because it is a chronic affair and cannot be cured by local measures. All of us recognize that some of these cases are dangerous and do not wait until a complication develops before we advise the patient accordingly.

As regards the question raised by Dr. Barnhill that a chronic running ear constitutes a possible focus for systemic infection, I have been very much interested in problems of focal infection and I have come to regard the ears with middle ear disease as rarely, if ever, constituting a focus for systemic trouble. Systemic infection is not prone to develop from infection of the mucosa, whether this is that lining the middle ear cavities, the wall of the pharynx, or the mucosa of the nasal accessory sinuses. Foci which do cause systemic trouble are to be found in buried infection, such as that which we encounter in the teeth, and in the depths of the tonsils. In this connection I would emphasize particularly infection in tonsil fossæ where remnants of tonsil structures have been imbedded in the scars following tonsillectomy.

Dr. George L. Tobey, Jr. (closing): I do not agree with Sir Charles Ballance that every chronic suppurative ear should have a radical mastoid operation after discharging 6 to 8 weeks, since we know conclusively that 70 to 90 per cent of our cases of chronic suppurative otitis media will clear up under conservative treatment. Four to five per cent of chronic suppurative ears really demand complete radical surgery, and a still smaller percentage develop intracranial complications. I have attempted to outline the indications for conservative treatment and its limitations. There is without question a half-way procedure between very conservative treatment and very radical operations, namely, middle ear surgery. Where there is evidence of intracranial involvement, infection of the internal ear, large cholesteatoma in the mastoid cavity, with evidence of an active suppurative process, a radical mastoid is of course indicated from the very first, and no time should be lost with any conservative treatment.

In answer to Dr. Barnhill, I agree with Dr. Shambaugh that in the presence of a perforation with a tympotympanic infection, a mucoid discharge with or without saprophytic bacteria, the case is not dangerous to the patient, and that it should readily clear up under conservative treatment. In the presence of a marginal perforation with cholesteatoma and evidence of active bone involvement in the mastoid, the radical procedure is indicated. Dr. Smith has laid particular stress upon the modified operation. I am yet to be convinced of its value or safety.

In speaking of radical operations he speaks of the preservation of the hearing as a very important factor. Radical mastoid operations are not performed to preserve hearing but primarily to prevent or to overcome intracranial complications. When indications for a radical mastoid are present, the complete surgical procedure should be carried out, in order that all vital areas may be under the direct observation of the otologist. As a matter of fact, in the average case we expect no loss of hearing, but a slight improvement.

Dr. Smith (closing): I agree with Dr. Tobey that the radical mastoid operation is primarily indicated not to improve the hearing, but to remove a dangerous necrosis in the temporal bone.

One of the more conservative operations will suffice in some cases while a complete radical operation will be indicated in others. In the absence of symptoms the problem depends largely on the type of discharge and its reaction to careful local treatment. When there is a mucous membrane discharge, or when the bone involvement is not deep seated or extensive, the reaction to local treatment is favorable. The foul odor disappears, the discharge becomes mucoid or stops entirely and operation is contra-indicated.

A continued foul smelling discharge persisting in spite of local treatment is indicative of deep seated necrosis within the mastoid and I believe the patient is safer with an operation. The choice of operation depends entirely on the degree of bone destruction. The first requisite of any operation is that it adequately removes the danger to the individual. This may be accomplished by one of the more conservative procedures in some instances, in others a complete radical mastoid operation will be necessary.

MORNING SESSION, WEDNESDAY, MAY 21, 1930.

The Physiology of Hearing.

PROF. ARNO B. LUCKHARDT, University of Chicago.

After reviewing the possible sense of audition in the various phyla, the author discussed hearing in the higher mammals, including man. The older and newer literature on the importance of the sound receiving, sound transmitting and sound conducting apparatus was discussed. The newer histologic researches of Held on the sound perceiving apparatus were detailed. The more important views as to peripheral tone analysis were outlined on the basis of the resonance and nonresonance theories of audition. The Hurst theory of audition was abandoned because of the recent experimental work of Kucharski (Pieron). The sound picture theory of Ewald was evaluated on the basis of histologic and physiologic evidence with that proposed originally by Helmholtz and later by Shambaugh. It was concluded that the resonance theories deserve adherence because of the available experimental evidence. The latest evidence (Held and Kleinmecht) favors the Helmholtz theory, namely, that the basilar membrane is the resonating structure.

DISCUSSION.

Dr. G. E. Shambaugh, Chicago: This problem of sound perception is rather complex, but there are certain fundamental facts on which we can all agree. The first of these is that the hair cells of Corti's organ are the anatomic structure in which the transference of physical to nerve impulse takes place. The second is that the stimulation of these hair cells is the result of some interaction between the projecting hairs and the membrana tectoria.

The fundamental problem about which there exists a difference of view is the manner in which this interaction between the membrana tectoria and the hair cells is brought about—whether it is the result of the vibrations of the membrana basilaris brushing the hair cells against the overhanging membrana tectoria, or whether it is the vibrations of the membrana tectoria itself which accomplishes this result.

There are two possible approaches to the solution of this problem. One is that of the physicist who, like Helmholtz, studies the phenomenon of sound perception and devises a possible theoretical explanation, and then seeks in the structure of Corti's organ the mechanism that seems to support his theory. Helmholtz accepted the idea that physical resonance was based on the stimulation of Corti's organ, but he looked upon the basilar membrane as the most plausible structure for filling this rôle of physical resonator. Vibrating in its several parts to tones of different pitch, it brought about stimulation of different groups of hair cells in different parts of the cochlea for the various tones of the scale.

The second method of approach to this problem is the biologic one—that is, to examine the structures in the cochlea in order to determine the probable rôle which each of these performs, and then to devise a theory that would suit these biologic considerations. Of the two methods of approach the biologic is certainly the more fundamental, and looking at the problem of sound perception from this point of view we can hardly escape the conclusion that the membrana tectoria is the activating structure which stimulates the hair cells.

The three types of end organs in the internal ear (the macula, the crista and Corti's organ) have the same fundamental construction—hair cells and the superimposed structure, namely, the otolith membrane, the cupola and the membrana tectoria. In the first of these, stimulation is unquestionably brought about by the movement of the superimposed structure, otolith, membrane and cupola. The logical conclusion is that in the third end organ—that in the cochlea, it is also the movement of the superimposed structure, the membrana tectoria, that responds to the sound impulse and stimulates the hair cells.

Our real problem therefore is to work out the probable response in the membrana tectoria which will best account for the phenomenon of sound perception. The explanation offered by Ewald has been rendered untenable by the discovery that the hair cells are normally in contact with and adherent to the membrana tectoria. Ewald's hypothesis was that the sound impulses produced undulations throughout the entire length of the cochlea and that only the hair cells occupying the crests of these undulations were brought in contact with the membrana tectoria and stimulated. Each tone in the scale, therefore, produces stimulation of a different grouping of hair cells, and this would account for peripheral tone analysis, which Ewald considered fundamental in any theory of sound perception. Now with the hair cells attached to the membrana tectoria, it is impossible to affect stimulation of separate groupings of hair cells by undulations passing through the cochlea, because every hair cell in Corti's organ must necessarily be stimulated and not only those which occupy the crests of waves.

I feel that we must always begin our speculations of the reactions in the cochlea with the biologic conception that it is the membrana tectoria that responds to sound impulses, and our problem is that of working out a hypothesis that will best explain how the vibrations of this structure account for the phenomenon of hearing. At present, physical resonance still seems to be the most likely method of response. Now, to say that the membrana tectoria cannot fill the rôle of resonator is hardly warranted, because we do not know enough about its structure to justify such a statement. Should, however, further studies of the structure

of the membrana tectoria warrant the conclusion that this structure cannot fill the rôle of a physical resonator we still cannot escape the biologic conclusion that it is the activating mechanism, and it will be necessary to work out some other hypothesis rather than that of physical resonator.

To my mind, most of the discussions one sees in the literature on theories of sound perception are off on the wrong foot, because it is assumed that the membrana basilaris must be the vibrating structure.

Dr. M. A. Goldstein, St. Louis: The description of eye witnesses to the Princeton experiment concerning the physiology of hearing has aroused me to a pitch of great expectancy. We have been talking and dreaming about theories of the physiology of hearing for over a half a century, and here is presented the first definite experiment on a living mammal which may afford opportunities to prove or disprove some of these theories, or, at any rate, to establish more definite data about this intricate problem.

If the pyramid of a living cat can be exposed by dissection and the auditory nerve hooked up with electric amplifying device, conducted to a soundproof room several hundred feet away, and the sounds of the human voice transmitted through the normal conducting apparatus of the cat's ear to the radio receiving apparatus in the soundproof room, a long step forward in this special physiological process will have been accomplished.

I wish to announce that through the courtesy of Professor Capps of the executive faculty, of Professor Langfeld of the department of psychology, and of Dr. E. G. Wever, conductor of this experiment in the laboratories university, as a mark of special courtesy to the Otologic Fellows, it has been arranged to perform this experiment at Princeton University Saturday morning, May 31st, and our Fellows are cordially invited to be present.

Dr. R. L. Wegel, New York: I take it that since Professor Luckhardt has been invited to give a paper on the physiology of hearing there would be a considerable practical use to otologists of a correct theory of hearing. Physiology ordinarily is concerned also with physics, so I will limit my remarks to that phase of the subject presented by the author. Professor Luckhardt has

designed his paper to give an account of the most noted theories and does not take sides with any one of them. To me the paper is a very important contribution, particularly at this time, because it shows in a very pointed manner the deplorable lack of agreement between these various theories. The lesson which I think we would do well to draw from this is that in view of the present intensity of effort in deafness research, it is quite time to find some way of intelligent study of the subject to discriminate between these theories and to develop a correct and therefore a useful one.

During the past fifteen or twenty years the science of acoustics has been reduced to a very good quantitative engineering art. When the theories described by Professor Luckhardt are viewed in terms of this recent experience their details can easily be seen to be hopelessly out of date and inaccurate. The standard procedure in developing these theories seems to have been to find a way of explaining a limited number of observed phenomena and usually to have insufficient regard for the physical possibility of the mechanism proposed. Careful examination often shows them to be violations of the simpler laws of nature.

In view of the shortness of the time allowed for a discussion, I would like to illustrate my attitude by picking one rather unimportant statement from the paper and discussing it briefly from the newer standpoint. The statement is that the ossicles are regarded as a bent lever system vibrating as a unit. This viewpoint is undoubtedly near the truth if these ossicles were to be used as a lever is used—that is, for exceedingly slow motions, such as might be encountered if the umbo were depressed with an instrument. It is, however, far from the truth when the motions to be considered are the vibrations resulting from sound. In this case they must vibrate with exceeding rapidity. The inertia of the ossicles then restrains their motion in increasing amounts as the frequency or pitch of the sound is increased. The malleoincudal joint is elastic and vibration must be transmitted through it. Such an elastic connection tends also to shunt or reduce the vibrations in increasing amounts as the frequency is increased. The lever action, therefore, granting that it is proper to speak of their action in such terms, changes considerably as the frequency

of the sound is changed, so that if we take for granted that the lever ratio of the system is 1 to 30, as Dr. Luckhardt says, for exceedingly slow motions, it may be almost anything else at sound frequencies. The action of the ossicles is further influenced in no small measure by the nature of the reaction of the apparatus at its two ends—that is, one end by the action of the membrana tympani with its load, the outer air and the connected tympanic cavities, and on the other end by the stapedius with its appended load, the labyrinth. It is not possible to give an accurate quantitative picture of this mechanism without a considerable excursion into mathematics, but it is possible in a general way to say that this part of the mechanism acts very much like a mechanical filter, which tends to suppress or to be insensitive to the very low frequencies (such as those for which the 1 to 30 ratio might be true), to favor the transmission of the most important audible range of frequencies—that is, perhaps, 300 to 3,000 cycles per second—and to discriminate against frequencies higher than this in amounts increasing with the frequency. This sort of result is in accordance with the determination of absolute sensitivity of the ear when properly interpreted.

The Princeton experiment mentioned by Professor Luckhardt has been cited as a possible argument against a mechanical theory of pitch discrimination and in favor of a central discrimination. I see no reason for changing the ideas which I have described qualitatively before you many times in regard to this subject. The end organ as a mechanism is capable of a certain amount of pitch discrimination regardless of how the nerves may function to sense the mechanical or sound disturbances to which it is subjected. I do not wish to say, however, that the burden of discrimination is left completely with the mechanism of the end organ. There is undoubtedly a kind of central analysis.

DR. PIERCE: Why?

DR. WEGEL: For one thing no mechanism seems to be capable of explaining the binaural sense. The eighth nerve branches after leaving each ear, one branch from each side passing over and connecting through centers with the other branch on the opposite side. Thus when an impulse originates in one ear it is transmitted to both sides of the brain. When impulses arise at both

ears, interference of the impulses transmitted to the brain centers is encountered on account of this cross-transmission, which interference varies with the time interval between stimulations on the two sides. In this general way a sense of direction is explained. Extremely low frequencies, such as are likely to be sensed as beat frequencies, may also to a degree be analyzed centrally.

DR. M. A. GOLDSTEIN, St. Louis: Dr. Wegel witnessed the Princeton experiment. I would like him to say what he saw.

DR. WEGEL: The auditory nerve of any anesthetized cat was exposed just beyond the point where it leaves the modiolus. Two electrodes are applied to the animal, one on this nerve, and the other at some other convenient grounding point which is itself not active. These electrodes are then made the input terminals of a vacuum tube amplifier, on the output end of which is a telephone receiver for listening. The set-up is the same as that usually used in the detection and recording of the so-called action currents except that a telephone receiver for listening replaces the conventional galvanometer for recording on film. When a sound is made in front of the cat, it is picked up by the end organ, which stimulates the nerve and sends impulses to the brain. These are picked up from the nerve on the way and are heard through the electrical apparatus. The striking thing about the experiment is that contrary to the usual conceptions regarding nerve transmission, extremely high frequencies are audible, and the conclusion is therefore arrived at that these frequencies are transmitted over the nerve trunk. It has been thought that the highest possible frequency of transmission was about 1,000 cycles, but in this case the Galton whistle could be heard, and the voice was very easily distinguishable. This experiment establishes the fact that frequencies higher than those permitted by the accepted length of the refractory phase are transmitted over the nerve trunk, but I believe it has not been established that such frequencies may be transmitted over a single fiber. Thus, if a single fiber was capable of transmitting only, say 1,000 impulses per second, ten fibers stimulated in the proper fashion could transmit 10,000 cycles per second if the sum total of the impulses were picked up by an amplifier.

Dr. J. Gordon Wilson, Chicago: We rarely have the pleasure of hearing a paper so clear and so judicious. Discussion is difcult. All I can hope to do is to consider some points of special interest to me as an otologist.

The method of the perception of vibrations either by tactile organs or by the ear is a problem fundamental to investigation of hearing. A mechanism to receive vibrations by air or water, a mechanism to protect the individual, is present in all forms of animal life. But a definite structure, from which the cochlea comes, appears late in the phylogenetic development in vertebrates. In the mammalian cochlea we have a highly specialized tactile mechanism. It may be that the peripheral organ of hearing like the labyrinth is not most highly developed in man. But when one considers not only the peripheral mechanism but its central connections there is no doubt it reaches its highest development in man, where sound is not only perceived but intellectually interpreted.

In fish with the budding cochlea (lagena) we may discern an evolutionary step of great importance. As one would expect in the transition stage, it is not easy to determine the method of perception, whether it is a tactile response from the lateral line organs or a response from the beginning cochlea. It has always seemed to me that fish respond to stimulations which are tactile shocks transmitted through a watery medium. There is evidence to show that the lateral line organs are necessary to orient fish to a source of vibration, to sense direction, though not essential to perceive vibrations. Here we may find a suggestion to help in the solution of the function of the saccule in mammals.

Higher in the vertebrate scale we have a more developed but primitive cochlea which perceives and discriminates sounds. These may be carried chiefly from ground vibrations, as in reptiles, or air vibrations, as in birds. In each we have the middle ear adapted to give the maximum aid, a good example of adaption to environment.

That the cochlea acts as a linear auditory analyzer seems to be generally accepted by physiologists and aurists. We all know that work of Wittmaack and Yoshi in which the destruction of

certain areas results in loss of the corresponding pitch. More recent work confirms this. Thus complete destruction of the organ of Corti on one side with partial destruction of the organ of Corti on the other, led only to the disappearance of conditioned reflexes to a certain range of vibration frequencies depending on the area of the cochlea damaged.

Physiology of hearing must not confine itself to the cochlea. All defects of hearing are not due to damage of the cochlea. As aurists we do well to concentrate on the peripheral organ. But there are times when one wishes he had thought of a central part. Some years ago I described in the Transactions of this Society (1916) a case of deficiency of hearing probably of central origin. In this case the range for forks was good, 24/16,000; the deficiency was of the nerve deafness type. Certain features in the case led me to send the boy to a psychologist who reported defective auditory association with inaccurate auditory memory, inability to associate sounds with words and other defects which we believed to be of central origin.

In the brain there is a relatively small cortical area of importance in hearing in the temporal lobe (Heschl's gyrus). According to present views it is the place where the incoming path reaches the cortex before its final redistribution. But note that the path has not arrived as it left the ear. Each has passed through junctions or the synapses where other relationships are probably established. What takes place at these junctions we do not know, but I do not think it a rash guess to say that some sounds passing through the cochlea are relayed at lower levels as unconscious reflexes. We have reason to believe that nonconscious reflexes are more active in lower mammals, e. g., the dog and monkey, and probably some of the unconscious auditory reflexes in man take place there.

Of what occurs in the temporal lobe to impulses sent in by the cochlea we have little knowledge. Attempts have been made to draw a parallel between the eye and the ear. One must be careful in drawing such a parallel even if we grant that nerve impulses are of a similar nature in both. The ear is a modern development compared with the eye. The individual afferent

fibers in man, which make up the visual path, end in the visual cortex as a solitary afferent, and their destruction is never recovered from. We have no evidence that individual afferent fibers from the cochlea impinge on individual points in the temporal lobe, though some claim that they do.

We may state this in another way. The projection of the retina upon the Calcarine area is a precise patterning of the impingement of visual afferents upon the cortex. The result is that removal of the projection areas is never recovered from. If it were so in the auditory area we should expect to find somewhat similar results. For instance, tone islands would exist not as we now conceive them, great depressions in the audiogram due to a faulty cochlea mechanism, but absolute losses.

Experimental removal of the cerebral cortex in animals gives very curious results. Many years ago I removed the left temporal lobe of a monkey to study the result on the vestibular reactions. We made no exact hearing tests but we noted that the monkey heard apparently as well as before the operation. This was no new observation. Professor Schafer and Dr. Sanger Brown of Chicago had removed both temporal lobes of monkeys and apparently with complete recovery of hearing. However there is so wide a gap between the monkey (even in as high a form as the chimpanzee) and man, that it would be rash to bridge the gap too hastily from experimental findings. Recently there has come to us evidence that makes it difficult to correlate the anatomic data in man as now taught with recent physiologic findings. I am referring to the results of Dr. Dandy in which audiometric charts were published by Dr. Bunch. Here after removal of the cortex of the right temporal lobe in man there was no evidence of a difference of hearing deficiency in the two ears. But when the lesion is bilateral the old teaching still stands. The faculty of hearing is disturbed in man by a lesion of a small area of the temporal lobe where the auditory path enters the cortex before its final dispersion into its association areas, and then only when the lesion is bilateral. No case has been recorded in which this narrow bilateral lesion has not seriously impaired hearing. This area cannot be regarded as a center of function

for so great a faculty as hearing, but rather as a dispersion area from which spring many association relationships, the intellectual spheres which are the real bond between minds.

DR. JACQUES HOLINGER, Chicago: Dr. Luscher in Bern has constructed a microscope for the ear with which to study the minutest movements of the drum head. He came to the conclusion that there is an accommodation movement on the part of the muscles of the middle ear and the chain of ossicles not only in listening, but at any time when a sound strikes the ear. In other words, the accommodation is constant and involuntary.

When the tensor tympani and stapedius muscles contract simultaneously, the chain of ossicles becomes rigid, and acts as a single lever, which on account of its fixation has no momentum.

In regard to the function of the inner ear, Nabuo Satoh's work on over-irritation of birds' labyrinths must not be overlooked. Corti's organ in birds (the lagena) is wide enough to study the action of the tectorial membrane. Birds were exposed to very loud explosions, and by special technic the parts of the inner ear were hardened in that position. It showed that the hair cells furtherest away from the insertion of the tectorial membrane were very widely thrown out of their position, while those nearest the insertion were intact. Therefore, function is produced by intercorrelation between the two. The only quesion is whether the tectorial membrane comes down on the hair cells or whether the hair cells come up against the tectorial membrane. Anyone who studies the carefully drawn pictures in Nabuo Satoh's atlas can hardly escape the conclusions that the tectorial membrane came down on the hair cells, since at the place where the tectorial membrane made the widest excursion the greatest effect on the hair cells is evident.

DR. WILLIAM J. MCNALLY, Montreal: I wish to thank you for giving me the opportunity to discuss this paper. I think we should be very much indebted to Prof. Luckhardt for this complete survey of the physiology of hearing. It is a very unbiased presentation of a very controversial subject.

The greater part of the literature on the physiology of the cochlea consists of argument without much experimentation. Owing to the difficulty of carrying out hearing tests on animals, it

has been almost impossible to do experiments on the cochlea of the living animal and therefore theorizing and experimentation with models have been resorted to. The American Otological Society has appreciated this difficulty and has established several research fellowships for the study of methods of testing hearing in animals.

There are two points in this paper I would like to refer to especially. The first point is in regard to the resonance theory of hearing. There are arguments for and against it, but on the whole, it is considered the most practical in meeting clinical requirements. If the resonance theory is admitted, it must be decided whether the basilar membrane or the tectorial membrane is the resonating element in the cochlea. I am not so unbiased as Prof. Luckhardt. I have been won over by the arguments of Wilkinson and Gray to the basilar membrane theory. Their main argument is based upon the fact that for a structure to resonate to a wide range of tones, it must vary its length, mass and tension. The basilar membrane is more likely to conform to these requirements. Both the basilar membrane and the tectorial membrane have fibers of varied length, shortest at the base of the cochlea and longest at the apex. The difference in length of the fibers will account for one and three-quarters octaves of our range of hearing. As to the mass or loading of the fibers, there is not much difference in the thickness of the fibers of either the basilar or tectorial membrane at the base or apex of the cochlea. The loading is probably done by the fluid in the cochlear chambers. The short fibers have a small fluid load, as they are at the base of the cochlea near the stapes and oval window. The long fibers at the top of the cochlea have a fluid load of the whole length of the scalæ. This loading is similar to the loading with copper wire of the long strings at the base of a piano. The variation in length of the fibers and the fluid load will together account for about four octaves of our range of hearing, and these two variable factors are common both to the basilar and tectorial membranes. However, there is a range of seven octaves of hearing still to be accounted for, which can be explained by variation in tension on the fibers. There is no evidence to show that there is any difference in

tension on the different fibers or sectors of the tectorial membrane, whereas Gray seems to give conclusive evidence of great variation in tension on the fibers of the basilar membrane. At the base of the cochlea, the spiral ligament to which the outer end of the fibers of the basilar membrane are attached is very large and here the basilar fibers are long. The arguments of Wilkinson and Gray show that the basilar membrane is best suited to act as a resonator for the eleven octaves of human hearing.

The second point I would like to emphasize in Prof. Luckhardt's paper is that apart from the fact that we cannot carry out hearing tests upon animals, it is possible that the cochlea is not the whole factor in the question of human hearing. Some of the other structures of the labyrinth might also play a part. I have been concerned with work on the saccule in the frog. When we cut the nerves to both saccules, we are unable to detect any change in the vestibular reactions of the frog. This has been confirmed by other workers on other species of animals. Most experimental evidence shows that the saccule is not concerned with equilibration. Parker showed that certain fish react to sound vibrations. The reactions disappear after ablation of both saccules. Von Frisch and deBurlet also studied the saccule in fish. deBurlet found the saccular macula is so modified in certain fish as to suggest a type of hearing apparatus. Sound vibrations might be conducted through the swim bladder, the perilymphatic spaces being connected to the saccular chambers in such a way that any sound wave in those spaces might affect the projecting saccular otoliths and maculæ and possibly act as a hearing mechanism. Von Frisch has been able to establish a conditioned reflex in certain fish so that they give a definite response to a particular tuning fork. The fish are taught to look for food when they hear a special sound. When he removes the semicircular canals and utricles, the animals still respond to any sound to which they are conditioned. When he removes the saccule and lagena of both sides, the animal loses its response to the high notes, i. e., above 64 vibrations per second. These experiments suggest that in the fish the saccule is part of the hearing mechanism, and in view of the fact that even in one of the mammals (rabbit) the saccule has no vestibular function,

it is possible that also in the mammal, the saccule will be shown to be part of the hearing apparatus, and may explain some of the things that at present are so difficult to understand with our cochlear theories alone.

The Value of Vestibular Tests in Intracranial Complications From Suppurative Diseases of the Ear.

DR. WELLS P. EAGLETON, Newark, N. J.: A great deal of what we have been taught to believe is so, is the result of theory that is not true. This paper was written in 1923 for the New York Academy of Medicine, but not presented until 1925 at the American Neurological Society. It was sent to the Archives of Neurology and Psychiatry, was accepted, but was lost. This is the 1930 edition of the 1923 paper. Some time ago the essayist came across three lectures by Eli Smith on the Evolution of Man, which were read with interest. He also read the work of Gaskow in 1917, and this paper is the result of trying to correlate biological and chemical facts.

This is the study of four brains which, at the time of the insult to the nervous tissue by hemorrhage or abscess, showed definite localizing vestibular manifestations. In a short time these all disappeared, but the sensation of increased intracranial pressure remained. It has been proven that tests of the vestibular apparatus are of great value, but have to be taken in conjunction with the history. The nearer to the time of the insult to the nervous tissue the greater is the disturbance in the vestibular reaction and the more definite the information obtained. The further away from the time the more misleading is the information. Particularly in ocular examination is it difficult to tell which eye muscle is affected.

DR. WELLS P. EAGLETON, closing discussion of his paper: I do not know how many thousands of people we have turned in the chair in our office. Of course, these tests are most valuable, but we are talking about the localizing value of these signs. What I want to emphasize is that except at the time of the insult to the nervous tissue they have little value in reference to localization.

Studies in Bone Conduction.

VERN O. KNUDSEN, Ph. D., University of California at Los Angeles, and ISAAC H. JONES, M. D., Los Angeles: Among recent new conceptions is the conviction that a true paracusis does not exist. More recently still is the feeling that we have had a wrong conception of increased bone conduction. There is such a thing as increased bone conduction; in a sound proof room, if a plug of wax or water occludes the right ear canal the Weber is lateralized to the right ear; the bone acuity of the right ear is increased ten decibels. In routine clinical cases, however, we have found that cases of stapes fixation and otitis media, chronic, catarrhal, which show increased bone conduction in the office do not show increased bone conduction when tested in a sound proof room. This makes it clear, that in making such tests we have mostly been testing the degree of noise in the room rather than the cochlea of the patient. Such clearer concepts of paracusis and increased bone conduction will make it possible not to require any special physiology of hearing, but they will come to be explained as facts of hearing which apply both to the normal and to the pathologic ear.

The effort of the authors has been—

(1) To determine the relative bone conduction sensitivity of the normal ear for tones of different pitch.

(2) To compare the bone conduction measurements in the office with similar measurements in a sound proof room, in both normal and pathologic cases.

The authors present a graphic curve of normal bone conduction, analogous to the Fletcher-Wegel curve for hearing by air conduction. In a tabulated series of four hundred pathologic cases it was found that a considerable number exhibited "increased" bone conduction when measured in the office. Twelve patients were selected and tested in the sound proof room, including otosclerosis, 4 cases; otitis media, chronic catarrhal, 5 cases; trauma of middle ear, 1 case; old cochlear defect with recent middle ear involvement, 1 case; otitis media, chronic purulent healed, plus cochlear defect, 1 case. Their charts are presented. None showed increased bone conduction in the sound proof room.

In the sound proof room the bone conduction tests can be made as high as a frequency of 8,000. This makes it possible to give a graphic comparison of the air conduction curve and the bone conduction curve which is helpful in diagnosis.

A description of a sound proof cabinet was presented with specifications for its construction in any average office. Allowing for the labor of an average carpenter the total cost is about $100.00 to construct such a sound proof room.

The meeting ended with a service in memory of Drs. Charles W. Richardson and S. MacCuen Smith.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting of Monday, March 3, 1930.

THE PRESIDENT, DR. SAMUEL SALINGER, IN THE CHAIR.

PRESENTATION OF PATIENTS: CASE HISTORIES.

"Brain Abscess."

DR. G. W. BOOT presented three patients who had been operated upon for brain abscess.

DR. T. C. GALLOWAY presented the following case:

This girl, aged twenty-three, was admitted to the Cook County Hospital three weeks ago with an ear which had discharged for five years. Two weeks ago there was a severe earache which soon subsided, but during the last few days recurred, accompanied by vomiting. On admission there was severe headache, marked rigidity of the neck and a slight Kernig. The knee jerks were approximately normal. The visual fields and discs were normal. The spinal fluid was under considerable pressure and there were 3,200 cells to the cmm.

A mastoid operation was performed. The lateral sinus was uncovered and found to be normally bluish. The dura over the tegmen antri was uncovered and seen to be thickened and grayish. There was no visible pulsation but this was easily felt.

There was a question as to whether the patient had a pia-arachnoiditis, a beginning encephalitis, a subdural abscess or localized meningitis which should be drained, or possibly brain abscess. There were no focal symptoms before operation. The reflexes were equal on both sides. There was no nystagmus, no ataxia and no asynergy.

Nothing further was done than a simple mastoid operation with wide dura exposure, and the patient made an uneventful recovery. At once the temperature, which had been 100.5° F., sub-

sided to normal and the ear discharge disappeared. Successive spinal fluid cell counts were 900, 50 and 6. The patient felt strong and was apparently well at the time of presentation.

Dr. Galloway recalled three patients from the County Hospital, shown three years ago, with similar meningeal symptoms and fluid findings, also treated successfully only by wide dural exposure, and expressed the opinion that patients who have no focal symptoms should be treated conservatively.

One knows of wonderful operations that have very properly been performed by masters of operative technic and diagnostic finesse, who have found definite localized lesions inside the dura which have led them to these operative procedures; but these should not lead the ordinary otologist into rash interference or invasion through the dura without very positive indications.

Dr. Charles F. Yerger presented the following case of brain abscess:

J. C., male, aged 8, with no previous history of ear disease, developed an acute suppurative otitis media (left) on April 1, 1929. A simple mastoid operation, without exposure of the dura, was performed on April 20, because of slight persistent tenderness over the mastoid, and the patient made a good recovery.

On May 29th, he complained of attacks of slight nausea and vomiting, and had a temperature of 99° F. The possibility of brain abscess suggested itself, but the more frequent condition of gastrointestinal upset in a child of his age was considered more likely. Treatment along this line was instituted with good results. A week later the patient again complained of nausea and vomiting, and again he was treated for gastrointestinal disturbance.

Nothing more was heard from the patient until June 13, when Dr. Yerger was called in as a consultant. The child then appeared to be very ill, was much emaciated and anemic, and the temperature was 102° F. There was marked stiffness of the neck, a positive Kernig and Brudzinski sign and anomia, the latter being considered pathognomonic of a left temporal abscess.

The spinal fluid was under increased pressure, slightly turbid, and the cell count was 3,000, 95 per cent of which were polymorphonuclears. No microorganisms were found on smear or culture.

A diagnosis of left temporosphenoidal lobe abscess, associated with a circumscribed meningitis, was made and operation was performed on June 17, 1929.

The previous mastoid wound was opened, making a radical mastoid operation, with complete removal of the tegmen antri and tympani. The exposed dura was apparently negative and the brain showed no pulsation. A longitudinal incision was made through the dura from before backward.

The brain appeared normal except for the absence of pulsation. A Gifford brain searcher was inserted over the region of the tegmen antri but no pus was found. It was withdrawn and reinserted over the region of the tegmen tympani, and creamy yellow pus flowed out in large amounts under considerable pressure.

A one-fourth inch nonperforated drainage tube was inserted into the abscess cavity and the surrounding area packed with iodoform gauze. When the drainage tube ceased to drain, it was removed and another inserted, a tube being kept in the abscess cavity for thirty-six days before being discontinued. No irrigation of the abscess was done. On two occasions aspiration was attempted through the drainage tube but was unsuccessful. Three or four times a medicine dropper full of a 2 per cent solution of mercurochrome was injected through the drainage tube into the abscess cavity, apparently with good results.

Dr. Yerger emphasized two features in this case which he considered factors for safety. The first was that one should not operate on an acute brain abscess until it has become firmly walled off or encapsulated, which requires from three to four weeks. Second, keep the drainage tube in place until the abscess cavity has become obliterated or until suppuration has ceased. In his opinion, it is less harmful to leave the drainage tube in place too long than to remove it too soon.

DISCUSSION.

DR. BOOT congratulated Dr. Galloway on his success, and said that he had on several occasions made a slight opening in the dura and found nothing inside. In one or two cases he had thought this was a good thing to do, as it might relieve the tension and prevent further necrosis. The patients went on to recovery without the formation of pus.

DR. NOAH SCHOOLMAN referred to the case of a child, aged 7, who was admitted to the hospital in coma and no history could be obtained. Sugar was found in the urine and there was a question whether the coma was due to glycosuria, but the child kept her hand to her head and moaned. Examination showed a chronic suppurative otitis media with aural polypus. Operation was decided upon. The mastoid was opened and a cholesteatoma was uncovered which filled the mastoid cavity and covered quite a large area of the cerebellum. The dura felt like parchment and had no granulations. Spinal puncture revealed an increased cell count and pus in the fluid. The fluid cleared up within three weeks and the child made a good recovery. She recently returned to the clinic with an otitis media of the opposite side, which was treated by paracentesis.

The speaker agreed with Dr. Galloway that it was well not to explore too far in such cases.

DR. J. HOLINGER cited a case he operated upon three years ago, in which he found the dura over the cerebellum under increased tension and incised it. The patient did well for a time and then developed septic attacks with chills and abnormal temperature. The sinus was laid bare extensively, and an extradural abscess was drained. Again he did well for a week. Then the temporal lobe was uncovered and the dura was found not pulsating. It was incised and several drops of pus were obtained. The prognosis at that time was bad. In the course of time the brain substance forced itself out of the incision of the dura. For some time (two weeks) this hernia baffled them but suddenly, within two days, it covered with good epidermis. The boy now has a hernia about the size of half a walnut but has no discomfort from it whatever and is doing well.

DR. E. W. HAGENS presented a paper entitled

"Decalcification of Temporal Bones."

(AUTHOR'S ABSTRACT.)

The author first considered the various solutions used for decalcifying the temporal bone. He advised a 1 to 2 per cent solution of nitric acid in 70 per cent alcohol. The fluid is changed to a fresh solution every several days until the bones are decalcified. About three to four months are found necessary for the process. To determine complete decalcification the author first described the chemical reaction of testing for phosphoric acid with ammonium molybdate and phenylhydrazine hydrochloride. The presence of phosphoric acid in the decalcifying fluid results in the production of the blue oxid of molybdenum, thus showing that decalcification is not complete. When decalcification is complete phosphoric acid is absent and the testing fluid turns a yellow orange color.

This method, however, is not entirely satisfactory in that (1) It is more delicate than is really necessary; (2) small bone areas may still be present and the test be negative; (3) a certain amount of time is necessary to perform the test, and (4) when phosphoric acid is present there is no way of knowing how much or how little undecalcified bone is still present. The X-ray method was therefore used, and the author found that the roentgenogram tells immediately whether a bone is partly or completely decalcified. This simple test avoids the need of long experience and judgment such as has always been found necessary.

DISCUSSION.

DR. IRWIN G. SPEISMAN said that in 1927 he visited Professor Kolmer in Vienna and found him X-raying all of his temporal bones during the process of decalcification. During the last year, at the University of Illinois, they have been doing the same thing. They use a solution of 5 per cent nitric acid for decalcification and the specimens are X-rayed every week. They have found three weeks quite sufficient for the decalcification of ordinary temporal bone.

Dr. Wells P. Eagleton, Newark, N. J., presented a paper entitled

"Unlocking the Petrous Pyramid."

(This paper is to appear in full in an early issue of the Archives of Oto-Laryngology.)

DISCUSSION.

Dr. Roy R. Grinker (by invitation) said he thought Dr. Eagleton's apparent lack of esteem for neurologists was due to the fact that he was a pretty good neurologist himself. He had demonstrated that his knowledge of neurologic anatomy is much better than that of the average neurologist. He has done in the field of brain abscess what Cushing has accomplished in tumor surgery, namely, the obliteration of outside specialists. This probably is a sign of advancement.

Several points in the essayist's remarks he thought were rather important. He spoke of quite early finding evidence of localized meningitis; that before a meningitis secondary to a suppurative otitis media becomes general it may give rise to bloody but not purulent fluid. The speaker found it very difficult to reconcile that with his knowledge of the wide open subarachnoid space where adhesions form with great difficulty when there is any degree of pressure. It has been his fortune to study several cases of generalized brain damage following otitic infection in which the general symptomatology has been a serous meningitis plus a toxic brain reaction. These patients had either died within the first few days following the infection, or if the source of the infection was relieved they had recovered. He wondered whether Dr. Eagleton's examples of exposure of the dura, or penetration of the dura, and later a free flow of fluid had not been examples of this serous toxic meningoencephalitis which would have cleared anyway, provided the source of infection had been treated.

He was interested in the frequency with which nasal infections give rise to brain conditions, and called attention to a recent article published in England which demonstrated how easily nasal fluids may infect the subarachnoid space. Experiments had shown that within two hours after dropping stained fluid into a rabbit's

nose it could be distinguished in the meninges at the base of its brain.

The essayist's description of the variations in the visual fields he thought remarkable. It seemed to him, from the study of certain rather similar transient, varying hemianopsias, due to a neighborhood lesion, a so-called retrolenticular internal capsule softening, where the optic fibers are not involved directly but by vascular reaction, that the visual fields become normal very rapidly. Edema thus can develop and can affect seriously the function of the optic fibers temporarily. In the essayist's description of the carotid artery, his decision to attempt only partial ligation seemed to Dr. Grinker quite reasonable, for it is frequently found that in the circle of Willis some of the vertebral arteries may be very narrow and not sufficient to allow compensatory circulation to take place, so that a permanent hemiplegia may result.

Dr. Grinker also had been impressed by the fact that otologists apparently do lumbar punctures without as much trepidation as neurologists have. If neurologists suspect intracranial pressure of any gravity, particularly of posterior fossa origin, they fear the result of a lumbar puncture, and he was surprised that the otologists encountered so little apparent trouble in doing punctures on cerebellar abscess cases. He had seen cases in which death occurred within a very few minutes following this procedure, and felt that a little more caution should be used in doing spinal punctures. In spite of the fact that the information gained is often very important he hesitates to do spinal punctures in these cases.

Dr. Albert Knapp said he never had attended a more interesting meeting. He described himself as the kind of a neurologist who looks after the reflexes, and when there is an infection of the middle fossa of the skull he looks for symptoms from the oculomotor nerve, if there is infiltration, or congestion or swelling in the organs of the middle fossa. He does not hope to find these himself but enlists the interest of the physicians and the hospital nurses, because the symptoms of the oculomotor nerves are transitory, sometimes lasting only a very few moments, perhaps only half a minute. This happens

very often in tumors, and he thought he was the first to emphasize these symptoms in tumors, but it happens much more frequently in abscess. He called attention to the work of McEwan on abscess of the temporal lobe, which showed that in a surprising number of cases of abscess there are changes in the pupils and also in the oculomotor nerves.

It had been stated that in some cases of inflammation of this region it was sufficient to open the skull to afford relief and he had had the same experience. He thought this might be due to the influence of the air as well as to the relief of pressure. Also in some other fields the influence of air has been surprising. It is known that in the presence of tuberculous disease of the peritoneum if the abdomen is opened the tuberculosis will heal. He had had the same experience in one case with cancer of the stomach, liver, glands and peritoneum. The inoperable growth subsided for three years after a tentative laparotomy. In diseases of the skull contents the symptoms often disappear following skull puncture in spite of the small opening of the skull, but only for a short time.

He had observed two cases in which an encephalitis started apparently with ear symptoms, and there are some more cases of this type in the French literature.

When there is an abscess of long standing in the brain he thought it was well to search for focal purulent infection in the body, especially in the lungs after cases of bronchiectasis.

Dr. Alfred Lewy thought that Dr. Eagleton might have the honor of being recorded as the first man to practice drainage of the cisterna pontis lateralis. When the speaker published his paper on this subject he found nothing in the literature. He reported some time ago the case of a boy who developed meningitis following a mastoid operation, and he enlarged the mastoid wound and went into the cisterna pontis. The boy recovered, but still has vertical nystagmus. There were no other neurologic symptoms that he could discover at this time.

Where a collection of fluid accumulates on the petrous pyramid, or even in the cisterna pontis lateralis, it is not necessary

to destroy the labyrinth to get at it. In his case he went right through, regardless of the posterior canal, but has since found that one can stop before reaching the posterior canal, and with an ordinary septal elevator can separate the dura from the petrous portion and break into the cisterna pontis lateralis and find the fluid. The operation may also disclose small abscesses in the posterior fossa in the region of the saccus. He had had no experience in going into the superior surface, as the essayist does, and did not believe the operation he had carried out would reach the marrow space in the tip as he described it.

Dr. J. Holinger stressed two points. First, it often is forgotten that the brain symptoms may change from day to day and even in a few hours. A physician may be called in consultation and fail to find symptoms that have been described as present. Symptoms may disappear and others appear in a very short time.

Second, he could not agree with Dr. Eagleton that the spaces medial to the labyrinth in the tip of the pyramid of the temporal bone are always marrow spaces. Siebenmann shows pictures of temporal bones that have been filled with Wood's metal by pouring the metal into the cells of the masoid. They showed also that there is a connection between the mastoid cells and the spaces in the tip of the pyramid. He thought that evidently both views were correct, that there are cases in which there is no connection and others in which there is a direct connection with the cells. This connection may be formed by a canal which goes along the edge of the pyramid. Regular air cells in the tip of the pyramid are an exception, if not a rarity.

Dr. E. W. Hagens said that in the specimens he had studied he had searched for pneumatic spaces in the front end of the petrous bone, but the only spaces he had seen so far had been marrow spaces, as Dr. Eagleton said. They are filled with marrow cells. He had, however, seen pneumatic spaces lateral to the eustachian tube region.

The speaker stressed the point that the cases studied were limited and he spoke only from his personal experience.

Dr. Wells P. Eagleton, in closing the discussion, said that he was not a pathologist and did not believe all the future of medicine was in pathology. When he studied the microscopic specimens in his three cases the sections of the petrous apex looked very different than those of the many mastoids he had seen. The petrous apex is developed from one center, which appeared to him to be the explanation of the findings. The specimens were different and it was also an amazing clinical fact that in examining necropsy material many cases show nothing but redness or discoloration of the bone, without caries. He said he would get a good pathologist to substantiate Dr. Hagens, for he knew these cases were different and that there are two kinds of bone in the temporal.

Replying to Dr. Holinger, he agreed that when dealing with infection the symptoms are associated with edema and change rapidly, but the edema of the brain is different from that of other parts because when it spreads it gives marked symptoms. A patient with an abscess of the temporal lobe at one moment will show symptoms, and the next moment will not. This is especially the case with the ocular field, because the findings are due to edema.

Regarding Siebenmann's work, he urged that anyone who could pick up a volume of his work on the temporal bone should buy it, no matter what it cost, for Siebenmann called attention to the paratubal cells, which the speaker believed were pneumatic. He believed they were developed from a different center than the labyrinth cells.

Dr. Lewy's cisterna pontis lateralis he considered a definite entity. It has not been described because investigators had not used a proper method—viz., dyes in solution by gravity—as the speaker had in his cadavers (Arch. of Surgery).

In reply to Dr. Knapp, in regard to the third nerve, the speaker said he considered it most important. It runs alongside in the arachnoid in a lake of its own, and any trouble, no matter how slight, may cause irregularity of the pupils. The pupils should be examined carefully in every case.

The motor portion of the oculomotor nerve does not seem to be very sensitive to pressure, but the sensory portion apparently responds to the slightest interference. The sympathetic fibers that come up in the carotid and go from there to the ciliary ganglion are extremely important, as was emphasized in one of the cases reported.

Replying to Dr. Grinker, the speaker said that he had nothing against neurologists, but as a class they are dealing with degenerative lesions, while he was dealing with suppurative lesions, and the same rules do not apply. His consulting neurologist takes his point of view, and believes that given a running ear and neurologic symptoms, the neurologic symptoms, unless they fit into something very definite, are apt to be associated with the suppuration in the ear. They see many cases with neurologic symptoms that do not go on to anything, but many do. He fully agreed that a routine neurologic examination should be made in every case; in fact, that there should be repeated neurologic examinations.

What he knew about meningitis he had acquired from cadavers. In eight cadavers he injected a pigmented fluid into the ears after opening the labyrinth, to find out where it would go, and this showed that the arachnoid pathways recorded in the books are wrong, for in the cadavers those illustrations in the books showing the large cisterna of the base and their free communications had been injected under great pressure. He found that the spaces do not communicate freely one with the other, but that the communications are divided by such fine trabeculæ that if one opens the mastoid and the labyrinth and fills the ear full of dye, if the dye is in solution it flows from that to all parts of the arachnoid base, but if the dye is in suspension it will go a certain distance and then meet a fine space and go no further. While cerebrospinal fluid can pass, the spaces are so fine that dye in suspension cannot pass, and that is what causes a localization of the meningitis.*

*Eagleton, Wells P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex. Arch. of Surgery, March, 1930, Vol. 20, pp. 386-420.

Referring to the experiment on rabbits that Dr. Grinker mentioned, he agreed that the findings were correct. Chemical dye is not an irritant and does not cause adhesions, but bacteria do, and for that reason the spaces are closed off, for Nature makes an effort to localize all infections.

Dr. Eagleton agreed concerning the danger of spinal puncture. He said he had seen deaths not infrequently, and had learned this: If there is a suspicion of increased intracranial pressure in the posterior fossa he does not do a lumbar puncture. If there is a suspicion of a blood stream infection, he knows he may cause a meningitis by doing a massive lumbar puncture and does not do it; although the withdrawal of a small amount for diagnosis is almost a routine.

In cases of meningitis they sometimes puncture and drain three times a day and he has never seen an accident in those cases.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, Held on Monday, April 7, 1930.

THE PRESIDENT, DR. SAMUEL SALINGER, PRESIDING.

PRESENTATION OF PATIENTS.

Rhinophyma.

DR. SAMUEL SALINGER presented a patient who had been operated upon for rhinophyma.

(ABSTRACT.)

Last fall I presented before this Society a case of an unusually large rhinophyma. I thought at the time it was the largest I would ever see. Today I present a similar case, which was even larger than the previous one.

One of the lobules which hung over the lower lip was so firm that I suspected malignant degeneration. Histologic section, however, proved otherwise. The operation was attended with comparatively little bleeding. Subsequently epithelial grafts were employed which helped materially in the cosmetic result.

DR. T. C. GALLOWAY presented a patient with primary carcinoma of the mastoid with cholesteatoma.

DR. PAUL R. CANNON, associate professor of pathology, University of Chicago, read a paper entitled

"Reticuloendothelial System as a Defensive Mechanism."

(AUTHOR'S ABSTRACT.)

The conception of a generalized system of cells of the connective tissues which are characterized by their frequency and intensity of phagocytosis was popularized by Aschoff under the name of the reticuloendothelial system. The designation of a so-called system is founded upon the monumental work of Metchnikoff, later amplified by many workers. With the advent of vital dyes, it became possible to show that colloidal and particulate materials are more readily removed by some cells of the connective tissues than by others, and that these cells are more

abundant in such organs as the spleen, liver, bone marrow, omentum, lymph nodes, suprarenals, hypophysis and to a lesser extent wherever loose connective tissue exists. This distribution may be demonstrated by injecting a suspension of particulate material such as India ink intravenously into an animal and finding the ink soon concentrated in the spleen, liver and bone marrow, and to a lesser degree in lymphoid and connective tissue. Practically none will be found in muscles or in tissues where the blood supply is rapid.

Bacteria are particulate particles when in the blood stream, and they also are removed principally by the phagocytes or macrophages of the spleen, liver, bone marrow, etc. In this way the body successfully defends itself against ordinary disseminations of infectious agents. The organisms are ingested and digested by the macrophages and the infection is terminated. All states of immunity are relative, however, and the effectiveness of the defense will depend upon the number and virulence of the organisms and probably also upon the functional state of the macrophages. At times the system of macrophages is inadequate to handle the infection and death may occur.

One of the problems of interest in connection with the property of phagocytosis of the macrophages is the possible relationship between it and the formation of immune bodies. Metchnikoff believed that the antibodies were merely products resulting from the digestion of protein substances by phagocytic cells, both microphages and macrophages. Later work has tended to minimize the importance of the microphages but has strengthened the view that the macrophages are the principal if not the sole formers of antibodies.

This belief has arisen because when organs richest in macrophages are removed, such as the spleen, the antibody production is significantly decreased. Also when the hemopoietic tissues are injured by chemical and physical agents, such as benzene, thorium-X and X-rays, the production of antibodies is diminished. Extracts of organs richest in macrophages also show the greatest concentration of antibodies. In tissue culture it has been shown that immune bodies are formed by macrophages. In recent times, experiments aiming to exclude a large number of macro-

phages from functional activity by means of so-called blockade have shown an inverse relationship between the concentration of antibodies in the blood stream and the degree of blockade of the phagocytic cells. All of these facts supplement one another and indicate that the macrophages are potent sources of antibodies.

The importance of these facts is apparent, inasmuch as it furnishes a means of determining the relative value of cellular and humoral factors in the mechanism of defense. When one compares the transitory state of passive immunization with the permanent degree of active immunity it becomes obvious that in the latter type antibodies in the blood stream must play a small part. And indeed in natural immunity there is little evidence that antibodies are of significant value. On the other hand, evidence indicates that in many infectious diseases the resistance depends primarily upon the numbers and functional activity of the macrophages, which lead to a relatively permanent active immunity. This does not exclude the participation of antibodies but suggests that they are of most importance intracellularly rather than in the blood stream.

Within recent years Besredka has written a book on the subject of local immunity in which he attempts to prove that immunity of local tissue may be achieved in the entire absence of antibodies in the blood, and, by implication, in the tissues themselves. His theories have been received with interest, but have not been confirmed by experimental facts. Nevertheless, Besredka has demonstrated that there is such a thing as local immunity of tissues, and has applied his ideas to the treatment of local infections of the skin and mucous membranes. His conception is that when bacteria are grown in broth an antagonistic substance is formed which is specific and gradually stops the growth of the organisms. To this hypothetical substance he has given the name anti-virus. By the use of broth dressings and injections of this anti-virus in tissues he believed that certain cells absorbed it and thus became immune to the bacteria later coming there. This anti-virus has had a wide clinical use in France and is now on the market in the United States.

Mr. Pacheco and I have been interested in this problem of local immunity and have performed experiments in an attempt

to analyze the mechanism. We have found that when a staphylococcus vaccine of killed organisms is injected intradermally for several injections and then the animal allowed to rest for three to four weeks, the later injection into this region of living virulent staphylococci is followed by a localized small abscess with no serious consequences to the host. The injection of a similar quantity of these living virulent staphylococci into the skin of a normal animal is followed by cellulitis and death in most cases. We have, then, a local immunity, produced by active immunization. In studying the tissues of such animals we find that whereas the staphylococci diffuse throughout the subcutaneous tissues in the normal animals and are vigorously phagocytized by polymorphonuclear leucocytes, nevertheless the infection generalizes and death occurs. On the other hand, in the immunized animal there has been a marked increase in the number of macrophages in the subcutaneous tissues, and these cells are extremely active in phagocytosis in the reinfection. In addition, there is evidence that in the immunized skin there is agglutination of staphylococci with a consequent fixation of the organisms at the site of inoculation, so that they are then more readily disposed of by the macrophages of that vicinity.

The local immunity seems to be due to two conditions: A local fixation of the bacteria, probably by a specific antibody present locally, and reinforced by the phagocytic activities of increased numbers of macrophages there present. This illustrates again a statement of Opie, that by this process "vital organs are protected at the expense of local injury."

DISCUSSION.

Dr. William Bloom, assistant professor of anatomy, University of Chicago, said several very important points could be gleaned from Dr. Cannon's presentation. He had shown clearly that if a large number of "reticuloendothelial" cells are put out of commission by India ink very few antibodies are liberated into the blood. He had also shown reaction of these cells on the reinjection of the inflammatory agent, and had reached a very definite, objective conclusion. Such work is in sharp contrast to the tremendous amount of speculative work which is written

about the "reticuloendothelial" system so freely, particularly in clinical literature. When the "reticuloendothelial" system is made responsible for everything which takes place in the body, it was refreshing to find that some objective, careful work could be done on these cells. Perhaps the best point made so far was their connection with inflammation—that is, the "local defense reaction." There was some evidence to indicate that these cells are involved in lipoid metabolism, but other than this he thought there was nothing to prove definitely that these cells participate in the metabolism of the body. This applied particularly to the part these cells might or might not play in any carcinomatous involvement. The big difficulty as Dr. Bloom saw it, and as he thought Dr. Cannon had found, was that in many of the processes which take place in the body one is dealing with antigens in solution, which are invisible. Until some way of demonstrating them with the microscope or otherwise is found it will be hard to link up the allergins with the "reticuloendothelial" system.

Dr. J. Holinger asked about the relations of these findings with Professor Bier's hyperemia. He pointed out that if one injects five to ten times a fatal dose of different kinds of poison or even of infectious material into the leg of a dog, after the leg has been tied up with an Esmarch bandage, the isolated leg will overcome the poison or infection and the dog develops no general symptoms. He asked if the manner in which this infection is overcome had anything to do with the liberation of antibodies.

Dr. Frank Novak referred to the cut which was presented showing an increased number of macrophages, and thought the mechanism of immunity might be explained on the basis of phagocytosis, but wished to know what connection the immune bodies have with it.

Dr. Alfred Lewy asked what evidence there was that the body fluids have to do with the bacteria, perhaps preliminary to their being ingested by the macrophages.

Dr. Budd C. Corbus expressed his interest in the subject and said that after reading Besredka's book it had occurred to him that he might accomplish something with the gonococcus. After great difficulty he had grown the gonococcus bouillon with ascites

fluid. He and his associate began their experiments first by injecting the urethra with gonococcal bouillon filtrate and sealing it in, but there was such a tremendous reaction to this that they had to stop the experiments. They then began to give intradermal injections, and they were surprised to find what a splendid response occurred with the proper dosage in clearing up the urethral discharge. They had treated some fifty or sixty cases before presenting a preliminary report to the Chicago Urological Society.

The first response noticed was an enormous exudate in which many intracellular cocci were found, later becoming extracellular. As the process goes on to extermination, the urine becomes clearer, there are a few threads showing a few polymorphonuclear cells, and finally these disappear and the patient recovers. They had given as high as 2 cc. They have taken the leucocyte count in all cases and found in some instances from 2,000 to 4,000 increase. In every case where there was a positive complement fixation test there was a strong response after the third week of treatment. They tested this out to see how long the antibody response persists.

Dr. Corbus said he attended the meeting because of his great interest in the subject of the reticuloendothelial cells, and expressed his gratitude to Dr. Bloom and Professor Cannon for their helpful presentation.

Dr. Paul R. Cannon (closing), referring to phagocytosis, said there is one fundamental feature, which is that phagocytosis probably cannot occur to any extent in circulating fluids. The very nature of the phenomenon demands a degree of quiet. It occurs mainly in organs that have a rather slow blood or lymph flow, as in the spleen, bone marrow, liver and lymph nodes. Where the current is stagnant and particles come in contact with a phagocytic cell there is an opportunity for them to be taken up. He suspected that the favorable effects of Bier's passive hyperemia in the example cited might be due to the fact that phagocytosis can occur where there is quiet, whereas under normal conditions many of the bacteria would be carried elsewhere. The mechanism of cellular immunity is also of importance in tuberculosis. The conceptions of the defense in tuberculosis have

been disturbed in the past year. It has been held that the reason a tuberculous reinfection remains localized is because there is an infiltration of cells which exert a barrier action and seal off the tubercle bacilli. Recent work has tended to indicate that this explanation may not be true. A very important principle has been developed by Opie in the demonstration of the mechanism of anaphylactic inflammation as a reaction to the meeting of antigen and antibody in the tissues. According to this explanation the cellular infiltration is secondary rather than primary, as usually believed. In the work on skin immunization just described there is definite evidence in the immune animals of an actual agglutination of the organisms *in vivo*, whereas in normal animals no such reaction occurs. He believed that antibodies play a very important part in the process. It is known that they are not the sole element, however.

Replying to Dr. Novak, Dr. Cannon said one interpretation is that phagocytic cells, once having had experience with a particular antigen, acquire an ability to digest it, so that later it may be digested with more rapidity. It is possible that this is due to the fact that tissues develop an ability to form antibodies more rapidly in later immunizations. There is much objective evidence supporting this view.

As to how the antibodies may act, Dr. Cannon said the current conception of antibodies in general is tending toward the view that many antibodies are of the same type, working in different ways, the agglutinating factor, the precipitating factor and the opsonizing factor all being different manifestations of one general force.

In regard to the work on the gonococcus, it is known that with most organisms the body can be specifically sensitized so that when the organisms are reinjected more reaction occurs than on the first occasion. It seemed to Dr. Cannon a logical treatment to use specific preparations in attempting to stimulate cells that are working against the gonococcus and are keeping it localized. By the specific irritation which is produced perhaps more of the cells may be mobilized and brought to the point of infection, and thus aid in phagocytizing the organisms. He believed there were

logical grounds for using such a treatment as Dr. Corbus had mentioned.

Dr. Samuel Feinberg, by invitation, read a paper on

"Nasal Allergy as Related to Hyperesthetic Rhinitis and Hay Fever."

(AUTHOR'S ABSTRACT.)

Hyperesthetic rhinitis and hay fever should be placed in the category of important diseases because of the frequency of their occurrence, the annoyance of their symptoms, the disability which they cause and the common occurrence of complications, such as sinus disease and asthma. There can be little doubt that the allergic concept explains satisfactorily a large proportion of the instances of hyperesthetic rhinitis. The author does not contend that this is the only possible theory nor that this concept explains all phases or all cases. The allergic concept presupposes a constitutional basis which allows particular individuals to become sensitized; of this basic constitutionality little is known. The immediate exciting agents in hyperesthetic rhinitis are the pollens, animal epidermal substances, foods, miscellaneous materials and possibly bacteria. Secondary etiologic factors are mechanical (dusts, polypi), thermal, neurogenic and infectious.

The diagnosis of hyperesthetic rhinitis can be complete only if the tests for the specific exciting causes are made. This is usually done by cutaneous tests, but at times also intracutaneous, nasal or conjunctival tests are indicated. There are many pitfalls in the interpretation of these tests, and they should be considered only in conjunction with clinical and therapeutic trials. Positive tests are obtained in about 99 per cent of seasonal hay fever cases and 30 to 50 per cent of perennial hyperesthetic rhinitis.

With proper attention to certain principles the treatment of the seasonal hay fever patient and the allergic type of hyperesthetic rhinitis is successful in the majority of instances. Treatment in the allergic cases aims at the elimination of the offending substances when feasible and desensitization when the former is not possible. Most of the failures are in the group of nonallergic cases. Here vaccine and local therapy have been the methods chiefly used. It is proposed that what we are now desig-

nating as hyperesthetic rhinitis may represent more than one clinical entity. It is possible that by closer coöperation between allergists and rhinologists greater progress could be made in this important field.

DISCUSSION.

Dr. Frank Novak said he was familiar with Dr. Feinberg's work and stressed two points, first, the necessity of drawing a very sharp line between the two conditions of hyperesthetic rhinitis and hay fever. They should not be used interchangeably, as they are two different conditions. Some cases of hyperesthetic rhinitis are allergic in nature, but the majority are nonallergic in nature. They have an endocrine basis.

Regarding the use of desensitizing procedures, Dr. Novak stressed the point that these measures should be carried out by a trained man who knows the subject of allergy, and not by one who has received his training from a representative of a pharmaceutical house, for the work is not innocuous. Some fatalities have been reported, even in the hands of experienced men. He believed the specialty should be practiced only by men who devote their time entirely to such work, and that others should refer their patients to such men for testing.

Dr. Leon Unger said he had worked with Dr. Feinberg for many years in this field and could corroborate his statements. He was sure that Dr. Feinberg treated the subject in a conservative manner and was not overenthusiastic, as many are. His figures in the results obtained in hyperesthetic rhinitis he believed were accurate. They have had a considerable percentage of good results and a considerable number of failures in the treatment of nasal allergy, but the longer they work the better are the results. They were becoming more and more convinced that skin tests must be corroborated by other methods of testing and that one cannot say a patient is not allergic because the skin tests are negative. A small amount of some irritant may have no effect upon a patient, whereas if a larger amount is used his threshold may be overstepped. The threshold idea he thought also explained the better results obtained when complete skin tests were made.

Dr. Unger stressed the point made by the essayist, that men specializing in sensitization work should work more in harmony with otolaryngologists.

Dr. T. C. Galloway emphasized the fact that many of the infectious conditions in the nose, especially sinusitis in children, are superimposed on an allergic state. He was surprised that the essayist gave such a low percentage of positives. They have obtained amazing results when they have in mind the possibility of an allergy in patients seen for obstinate infection. A typical case was seen first with otitis media and was seen with disabling infections every few weeks over a long period with the mucous membranes red and inflamed every time. Finally in a clear interval the characteristic pale, boggy membranes of allergy were noted, and although two sets of skin tests were negative, trial determined a sensitiveness to egg. When the allergic factor was removed the child's infections stopped completely.

Dr. Galloway thought the profession would hear more and more of allergy all the time, and that the Society was very fortunate in having such a scientific paper presented.

Dr. Walter H. Theobald complimented the essayist and expressed the opinion that an allergist must be a Sherlock Holmes. He was glad Dr. Feinberg had brought up the neurogenic type of case. Many such cases under their observation show negative skin tests but have symptoms of hyperesthetic rhinitis occurring any time of year. In his experience these cases are greatly improved and many times cured by cauterization of the so-called Killian sensitive points. Dr. Norval H. Pierce has reported many such cases in which there have been no allergic findings.

Dr. Samuel Feinberg (closing) expressed his appreciation of the discussion, and said that many patients with hay fever also have a hyperesthetic rhinitis lasting throughout the year. The men doing this work all the time see many patients come in with a diagnosis of hyperesthetic rhinitis and discover very marked positive pollen tests. Then the patient often recalls that his symptoms are more pronounced at certain seasons of the year, particularly those associated with pollens.

Dr. Feinberg was impressed with the idea that there is some association of asthma with hyperesthetic rhinitis, and of hay fever with hyperesthetic rhinitis.

He was glad the point about positive skin tests was appreciated, for if one depends on skin tests alone for a definite diagnosis many failures will result. Many tests should be carried out and the results correlated with the clinical findings and trials. He thought Dr. Galloway had misunderstood him in regard to the percentages cited, as he had referred only to the hay fever group.

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